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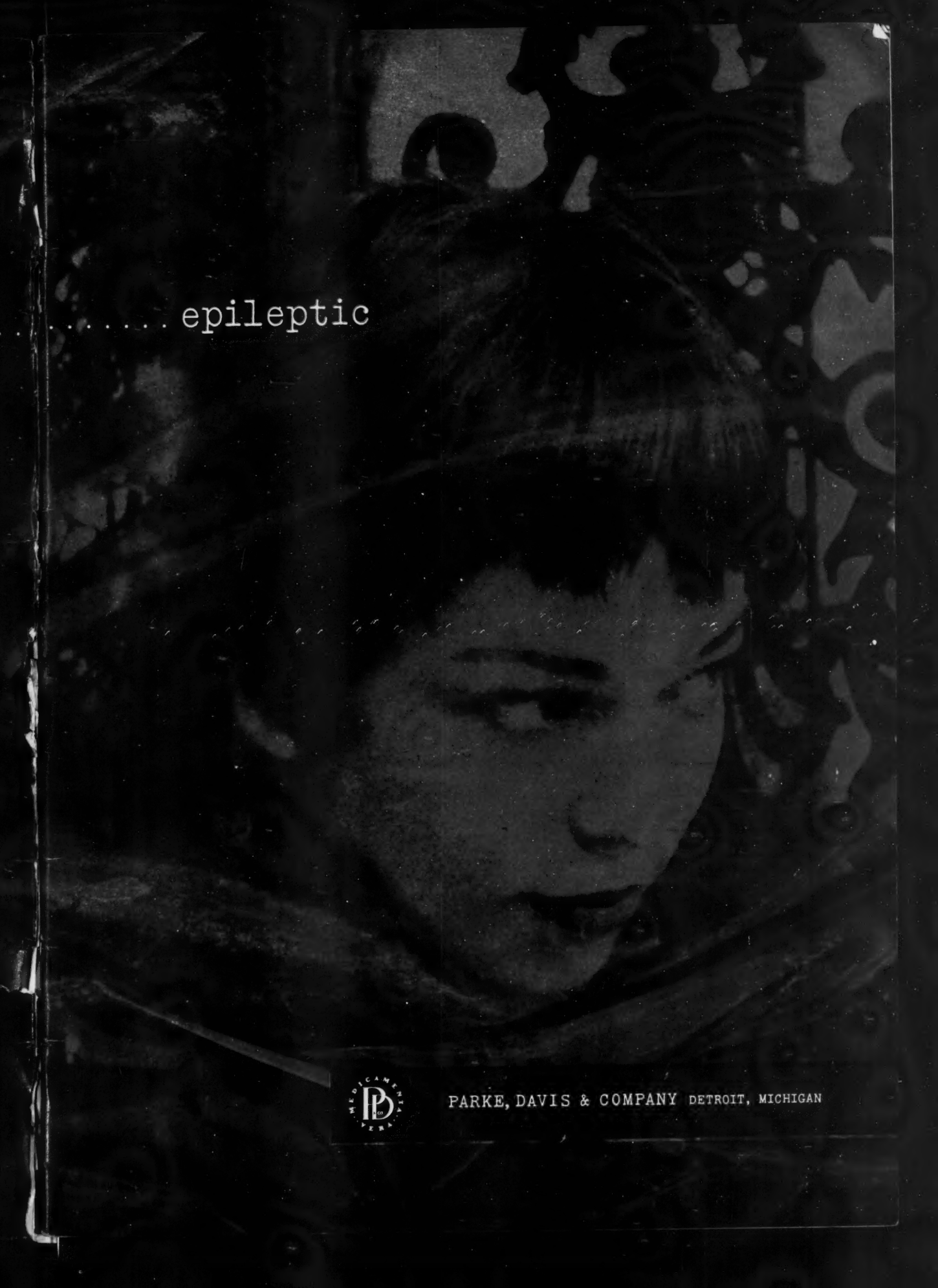

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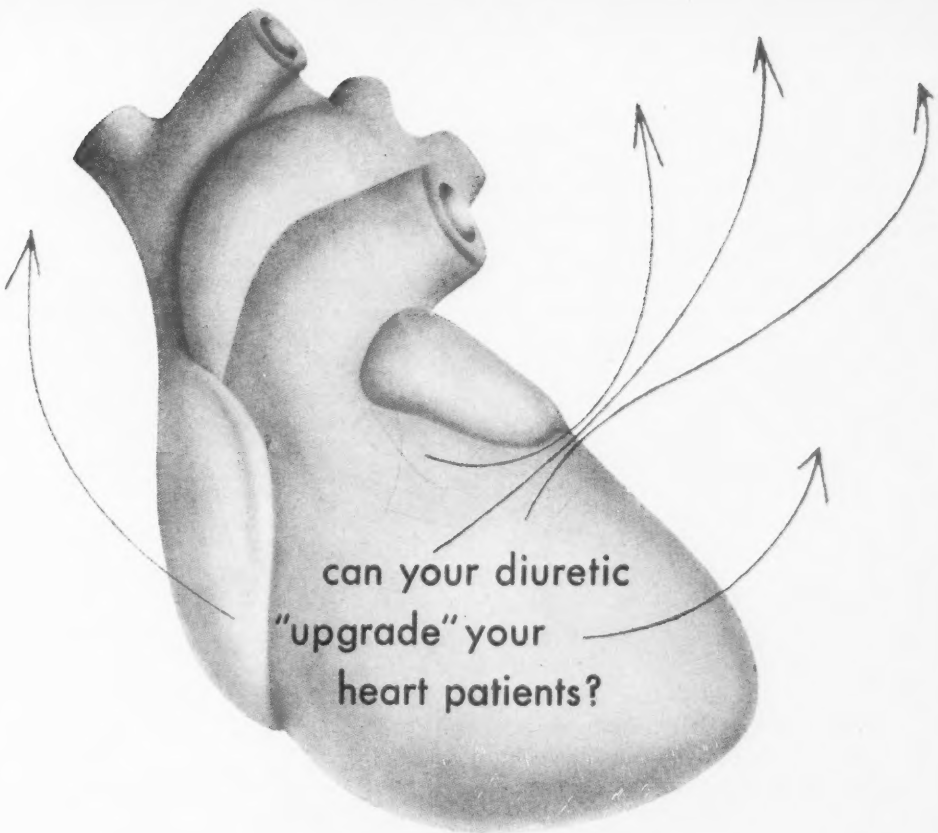

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Cooked Pork Chops, Ham, and Pork Sausage
Nutrients and Calories Provided by 3-Ounce Portions

TABLE I	Protein Gm.	Thiamine mg.	Niacin mg.	Riboflavin mg.	Iron mg.	Phosphorus mg.	Calories
Pork Chops, without bone, cooked, 3 oz.[2]	20	0.71	4.3	0.20	2.6	200	284
Ham, without bone, cooked, 3 oz.[2]	20	0.45	4.0	0.20	2.6	202	338
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TABLE II
Nutrients and Calories of Cooked Pork Chops (3 ounces) Expressed as Percentages of Recommended Daily Dietary Allowances[8]

Percentages of Allowances for:	Protein	Thiamine	Niacin	Riboflavin	Iron	Phosphorus	Calories
Girls, 13-15 years of age; weight, 108 lb.; height, 63 inches.	25%	55%	33%	10%	17%	15%	11%
Women, 25 years of age; weight, 121 lb.; height, 62 inches.	31%	59%	36%	14%	22%	17%	12%
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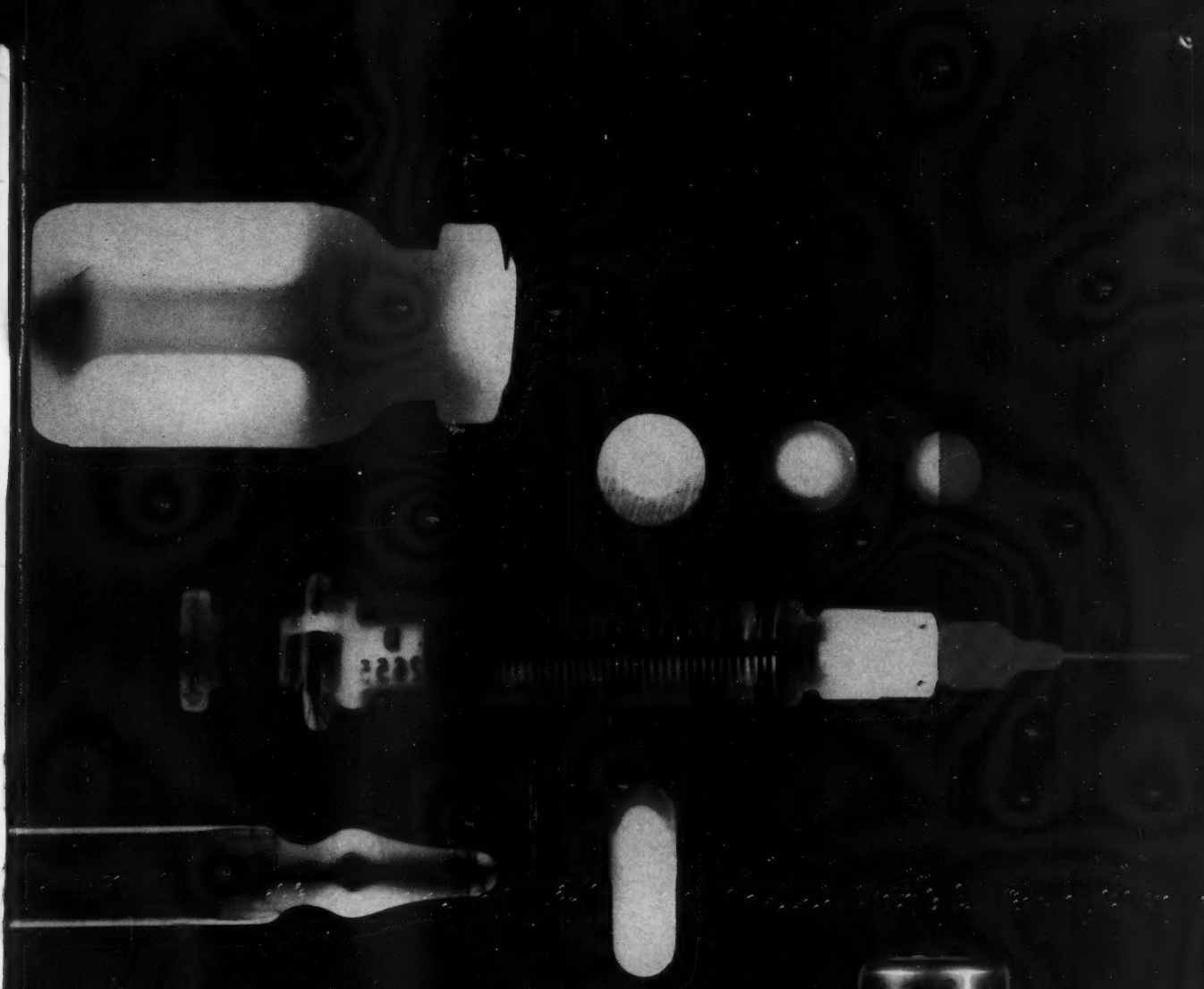

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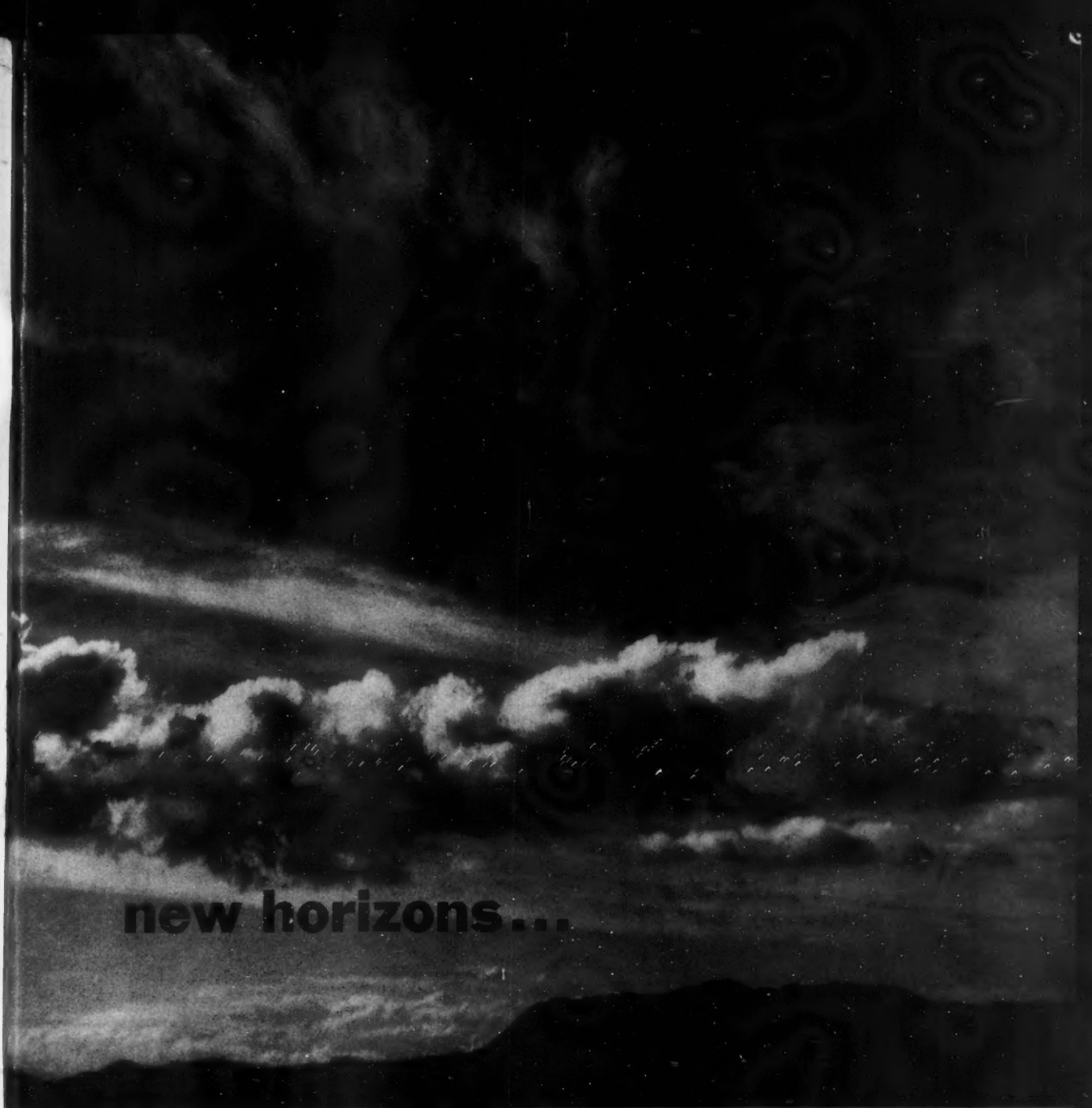

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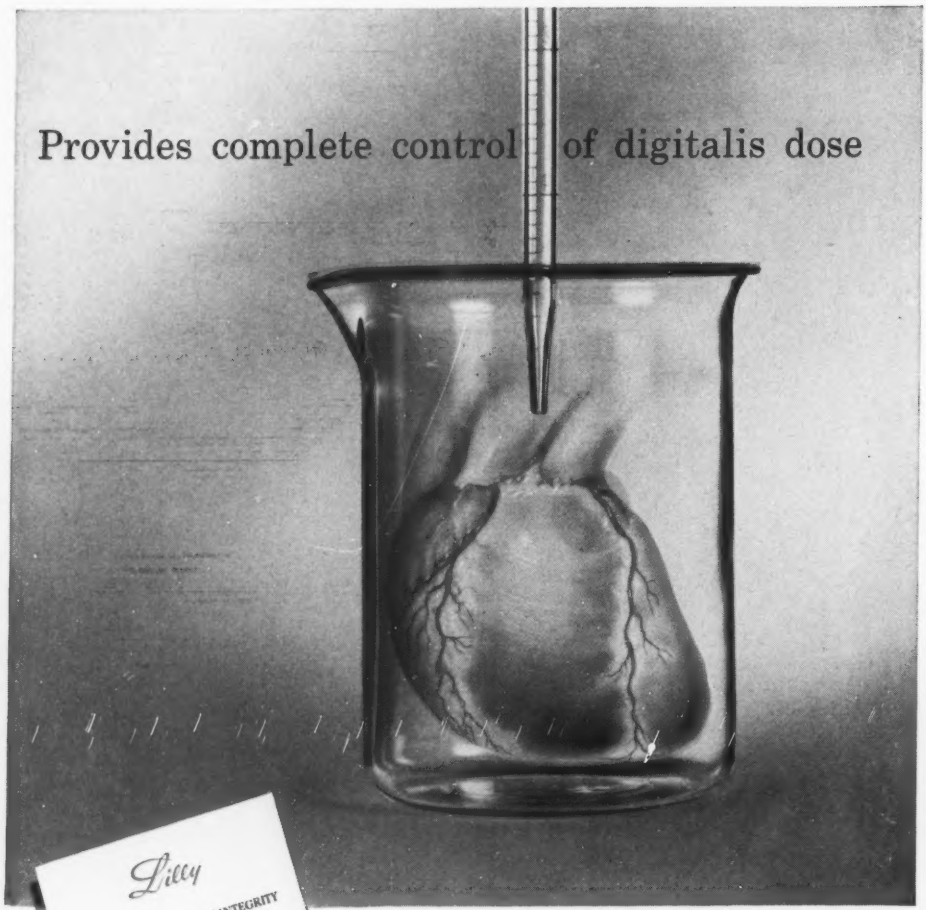

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Volume LVII **JANUARY, 1956** **No. 1**

Asthma

The Use of Khellin in Its Treatment

JOHN K. FULTON, M.D., ***Wichita***

Since the original publication by Anrep[1] in 1947 on the pharmacologic properties of the alkaloid khellin (visammin), reports have been controversial concerning the use of this drug in angina pectoris and bronchial asthma. Under laboratory conditions it has both coronary vasodilator and bronchodilator properties. However, the drug often produces severe nausea when taken orally and is moderately cumulative in action.

Initial results as reported by Major[2] were impressive in a series of 12 patients. Rosenman[3] studied 21 patients, nine of whom showed prompt relief of asthma attacks after a single intramuscular injection of khellin. More impressive perhaps was the effect he observed in treating eight patients with chronic cor pulmonale, all of whom obtained a response described as "excellent." In three of these, the clinical response was accompanied by rapid loss of edema, which previously had been refractory to digitalis and diuretics.

Employing pulmonary function measurements, including maximum breathing capacity and vital capacity measurements in asthmatic patients before and after oral and intramuscular administration of khellin, Snider[4] concluded that no significant bronchodilator effect could be demonstrated; however, in three of seven patients treated by the oral route there was a 20 per cent improvement in maximum breathing capacity, a difference they did not feel to be significant.

Silber[5] noted no benefit from intramuscular khellin in six patients with chronic pulmonary insufficiency without asthma. With oral khellin, which may have a cumulative effect, he noted an increase in maximum breathing capacity without consistent change in oxygen uptake, oxygen removal, cardiac output, or pulmonary arterial pressure. Cash and Zimmerman[6] were unable to demonstrate any change in pulmonary arterial pressure or cardiac output in five patients with pulmonary hypertension given intramuscular and oral khellin.

Tuft[7] injected 50 to 100 mgs. of khellin intramuscularly and reported appreciable degrees of benefit in 28 of 37 patients with chronic bronchial asthma who received more than one injection of crystalline khellin. Kenawy[8] noted complete relief of acute attacks in 102 of 138 patients and partial relief in 24 following intramuscular khellin.

It would seem from the foregoing controversial data that khellin lacks the reliability and freedom from toxic effects which would make it desirable in the routine treatment of bronchial asthma. The author's experience would tend to indicate that for the average case of bronchial asthma, ephedrine, aminophylline, epinephrine, iodides, and isuprel are far

SUMMARY

Khellin (visammin) possesses distinct value in certain cases of bronchial asthma not responding to other forms of therapy.

Response to this drug is unreliable and gastrointestinal side effects are common. It is not recommended for routine treatment of chronic asthma.

The suggestion is made that khellin acts through an unknown mechanism to relieve certain types of asthma.

From the Department of Internal Medicine, Wichita Clinic, Wichita, Kansas.

more practical and dependable in the symptomatic treatment of asthma. In the usual severe case, cortisone and its analogues are entirely adequate within safe dosage ranges for prolonged administration.

There remains, however, a small but troublesome group who do not get complete or adequate relief from cortisone in dosages considered safe or desirable for prolonged administration. There are a few patients who seem refractory to cortisone or corticotropin in any dosage, particularly those with long-standing asthma and associated emphysema. The cases presented below were selected because these patients were not amenable to any other known type of therapy and appeared to be examples of the employment of khellin where it was not only useful but actually essential to the favorable outcome. These case studies were not "controlled" except that in each case failure of previous treatment led to a successful trial with khellin and reluctance on the part of the patient to discontinue its use.

CASE I

A 51-year-old white male had begun to have chronic cough during adolescence which developed into distinct asthma at the age of 41. This was complicated by suppurative bronchitis and the development of severe pulmonary emphysema. Scratch tests performed in 1952 were entirely negative. At about that time he first developed signs of cor pulmonale with hepatomegaly, ankle edema, moist pulmonary rales, marked cyanosis, and secondary polycythemia. The hematocrit was 62 per cent.

Response to repeated intravenous infusions of ACTH and to oral cortisone was poor. The edema became progressive and extended to the waist line, associated with massive hydrops of the scrotum. The patient was extremely cyanotic, drowsy, and apparently terminal. He was then given khellin in a dosage of 40 mg. orally four times daily. In three weeks a marked diuresis occurred with disappearance of most of the peripheral and pulmonary edema so that ambulation became possible. Improvement has been slowly progressive in the past two and one-half years, and the hematocrit is now 51. The patient is able to walk two city blocks without difficulty. There is no cyanosis. There is occasional slight wheezing which is relieved by isuprel inhalations.

Summary: This patient had failed to respond to cortisone, aminophylline, intravenous ACTH, potassium iodide, and isuprel, as well as to a low sodium diet, digitalis, and mercurial diuretics. Relief was prompt and lasting with oral khellin in a dosage of 40 mg. four times a day which he has continued for two and one-half years.

CASE II

A 47-year-old man had onset of nasal polyposis at the age of 41 followed by cough, wheezing, and eventually attacks of severe intractable asthma which were not relieved by epinephrine. ACTH in an intramuscular dosage of 20 mg. every six hours gave only partial relief. Attacks were consistently produced by the ingestion of aspirin. He was then given cortisone in a dosage of 200 mg. daily which was later reduced to 75 mg. He was unable to obtain complete relief with the latter dosage.

After moving to Tennessee in 1951, he received desensitization treatments for six weeks for bacteria and dust without relief. He then visited an asthma clinic in Mississippi in October of 1952 and continued its medication until January of 1953. During this period he also saw two different psychiatrists, received a course of roentgen therapy to the chest, and had six nasal polypectomies in the two-year interval. His longest period of relief without cortisone was two and one-half months.

When he returned in 1954, he required frequent daily injections of epinephrine and had lost two good jobs because of his chronic illness. He was then given khellin in a total dosage of 240 mg. per day. He has now received this medication for one and one-half years. In addition, he takes 25 mg. of cortisone two or three times daily. An attempt to reduce the dose of either khellin or of cortisone resulted in a slight increase of symptoms. He has lost no time from work in the past one and one-half years. Very small nasal polypi were present on his last examination.

Summary: An aspirin sensitive asthmatic, not controlled by 75 mg. of cortisone or by other specific or non-specific measures, was rendered essentially symptom-free by the combination of khellin and cortisone in moderate dosage.

CASE III

A 61-year-old white male had had asthma since World War I. He had had a period of partial relief for a few months following a visit to an asthma clinic in Mississippi. The asthma was non-seasonal and had not responded to desensitization therapy administered elsewhere. The asthma did not improve on an elimination diet, and relief had been only partial from Meticorten in a dosage of 10 mg. four times a day. He was given khellin in a dosage of 40 mg. four times a day and has continued it for a period of four months. He has had no attacks of asthma since beginning khellin therapy. He no longer uses either epinephrine or other symptomatic medications.

Summary: A patient with moderate, chronic, bronchial asthma was unrelieved by hyposensitization, food elimination, Gay treatment, and Meticorten. He obtained complete relief with oral khellin alone.

CASE IV

A 48-year-old white male had had asthma for five

years at any time of the year. Attacks were not characterized by severe paroxysms, but he considered himself mildly asthmatic at all times. Physical examination on numerous occasions confirmed this impression. Treatment with cortisone in a dosage of 25 mg. four times a day, ephedrine, iodine, aminophylline, and intramuscular ACTH did not relieve his attacks. He was then given khellin in a dosage of 40 mg. four times a day, which he has continued to take for the past four months.

Summary: Mild chronic, intrinsic, bronchial asthma was unrelieved by cortisone, ephedrine, and iodine. Complete relief occurred with khellin alone.

CASE V

A 49-year-old white male began to have asthma at age 43. He had been unrelieved by iodine, ephedrine, and mold desensitization. Relief was definite with khellin for a period of one and one-half years. There was then a gradual return of asthma which responded promptly to cortisone in a dosage of 75 mg. per day, but returned on a dosage of 50 mg. daily. He then consented to take both khellin and cortisone in dosages of 160 mgs. and 50 mgs. per day, respectively. On this regimen, he has been essentially free of asthma for eight months.

Summary: Mild, recurrent asthmatic attacks were controlled on combined khellin and cortisone therapy. The use of khellin permitted a reduction in cortisone dosage.

CASE VI

A 29-year-old white male had had asthma for one year. Attacks were possibly precipitated by aspirin but were non-seasonal and often severe. Scratch and intradermal skin tests to foods and inhalants were not informative. He obtained moderate improvement with potassium iodide, but he had nightly attacks of asthma requiring epinephrine. He was then given khellin in a dosage of 40 mg. four times a day. Relief was prompt and has lasted 18 months.

Summary: A patient with bronchial asthma and possible aspirin sensitivity had sustained relief with khellin alone.

COMMENT

Several tentative impressions were formed from the results in these cases and in others where the drug was used but seemed to make no useful contribution to therapy. The drug seemed most valuable in patients with longstanding, chronic disease, was in all of the most strikingly successful cases employed in the male sex, and was beneficial in cases commonly considered to be of the intrinsic type. Five of the six benefited patients were past 40, and most of these had noted onset of asthma after the age of 40. Obviously this series is too small to justify definite conclusions. These cases are presented as a suggestion of the probable sphere of usefulness of this drug in other cases.

The irregularity of clinical response to khellin suggests that its mechanism of action may be other than that of a simple bronchodilator. It has been useful where conventional bronchodilators and anti-inflammatory agents, such as cortisone, have been ineffective. This suggests that khellin acts by a different mechanism to relieve asthma.

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The Telephone Electrocardiograph

The Practical Application of an Ingenious Device

GEORGE W. JACKSON, M.D., ***Topeka,*** **C. F. TAYLOR, M.D.,** ***Norton,*** **and J. L. MORGAN, M.D.,** ***Emporia***

The transmission of electrocardiograms over standard telephone wires has been widely described by the lay press as an eye catching and interesting development in the field of medicine. We would like to report our application of this principle as a practical and valuable time saving device connecting the Kansas State Tuberculosis Sanatorium in Norton with its consultant in cardiology in Emporia, 307 road miles east of the sanatorium.

The idea of sending electrocardiograms over telephone wires is not a new one. It has recently been recalled that William Einthoven used this method some 50 years ago to transmit electrocardiograms from in-patients in the hospital to his non-portable string galvanometer in the laboratory building located some distance from the hospital.[6] Einthoven called his remotely obtained tracings "telecardiograms." The presently used device has been termed the "Tele-EKG."[4]

The practicality of sending electrocardiograms instantaneously from the bedside via telephone wires to a *distant* consultant was established in this country by Dimond at the University of Kansas[1, 2, 3, 4] and Rahm and associates at the University of Nebraska in 1952.[8, 9] They incorporated the use of a small preamplifier which is connected to the patient and converts the electrocardiographic voltages into a frequency modulated signal. This is transmitted over the telephone lines and is demodulated at the receiving end where it can be recorded on tape, viewed on the cathode ray screen, or recorded on any standard electrocardiograph.

The applicability of this system is not confined to the transmission of electrocardiograms alone, since electroencephalograms can be similarly sent.[7] The frequency modulation feature has been carried a step beyond the telephone limitations and has even been adapted to the wireless transmission of electrocardiograms from an airplane to the ground[5]—though this obviously seems to be of experimental interest.

In October, 1952, we began investigating the feasibility of a Tele-EKG system for the Kansas State Tuberculosis Sanatorium. This institution, located at Norton, a town of 3,000 inhabitants, has 500 beds which are occupied in their entirety much of the time. The problem of adequately staffing the sanatorium with physicians is no different from that problem in all similar institutions, in that there is a rather frequent turnover of at least half of the staff physicians. From time to time able cardiologists have been on location at the sanatorium; however, there were gaps in the continuity of this coverage, so a consultant was appointed several years ago to insure a long time follow-up of the cardiac problems.

As a part of this service, electrocardiograms were sent by mail to Emporia, taking a minimum of four days in the mail for the round trip. Many of these were not of an emergency nature, but a sufficient number were of immediate interest to prompt the time saving consideration of the Tele-EKG. Finally, on February 8, 1954, the Southwestern Bell Telephone Company gave approval for the use of their lines for our transmission circuit, and on April 15,

SUMMARY

The telephone electrocardiograph has been an accurate and practical diagnostic device as used between the State Tuberculosis Sanatorium at Norton, Kansas, and Emporia, Kansas, over a period of a year and a half.

Figure 1. The sending unit of the telephone electrocardiograph.

Dr. Jackson is Director of Institutional Management, Kansas State Department of Social Welfare; Dr. Taylor is superintendent of the Kansas State Tuberculosis Sanatoria; Dr. Morgan is consultant in cardiology, Kansas State Tuberculosis Sanatoria.

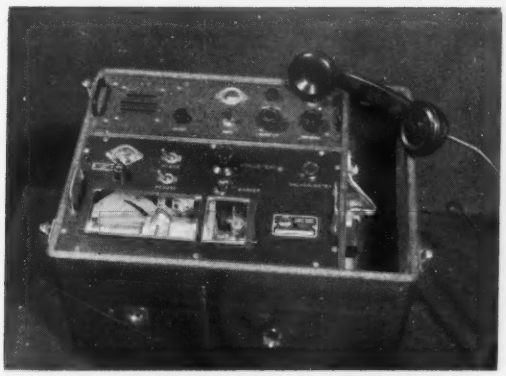

Figure 2. The receiving unit of the telephone electrocardiograph.

1954, we installed the first Tele-EKG made commercially following the design of Doctor Dimond's experimental apparatus.*

The actual operation of the apparatus is easily mastered. One technician from the sanatorium and one from Emporia were instructed in one day's time to respectively send and receive the Tele-EKG (See Figures 1 and 2). The expense of operating the Tele-EKG, once installed, is reasonable. The telephone rates are standard for station to station calls. For the Norton-Emporia connection, which covers 229 air miles which is the way telephone mileage is calculated, the rate is a dollar for the first three minutes and thirty cents for each succeeding minute until 16 minutes, after which time the rate is even less. It takes about three minutes per tracing, so this is approximately $1.00 per patient tracing.

Since April, 1954, we have transmitted electrocardiograms at least once weekly, and we have had no technical interference from the apparatus. The tracings recorded in Norton and those simultaneously recorded in Emporia have been uniformly identical, as illustrated in Figure 3.

The initial patients were studied in consecutive order to develop our technic, hence the diagnostic value of many of these early tracings was not of great immediate value. After the first month of operation, we limited our choice of patients to the following categories, a plan which we still follow:

1. Recent arrhythmias.
2. Chest pain suggesting heart disease.
3. Preoperative appraisal for *emergency* surgery.
4. Questions of electrolyte metabolism, particularly involving potassium levels.

We have a scheduled time of day (11:00 A.M.) to transmit though we can send and receive tracings at any time. If the consultant is out of the office when a tracing is received by the technician, he can be located and telephone the interpretation back to the sanatorium usually well within the hour.

There are some obvious limitations to this long distance type of consultation which we realized at the outset, and as we have gained experience we have learned some unanticipated lessons. Many times it would have been of great value to have previous tracings available for comparison, though our ingenious friends in the electronics field had already anticipated this. They state that electrocardiograms can be transcribed on steel tape and "played back" over the telephone whenever we desire.[7, 9] A present disadvantage is that a patient must be moved from his home or hospital room to the telephone where the Tele-EKG has been "coupled in." In time it may be that a practical means of "plugging in" to standard telephone equipment may be feasible.

* Made by Johnnie Walker Surgical Service Company, Kansas City, Missouri, and called the Tel-E-EKG.

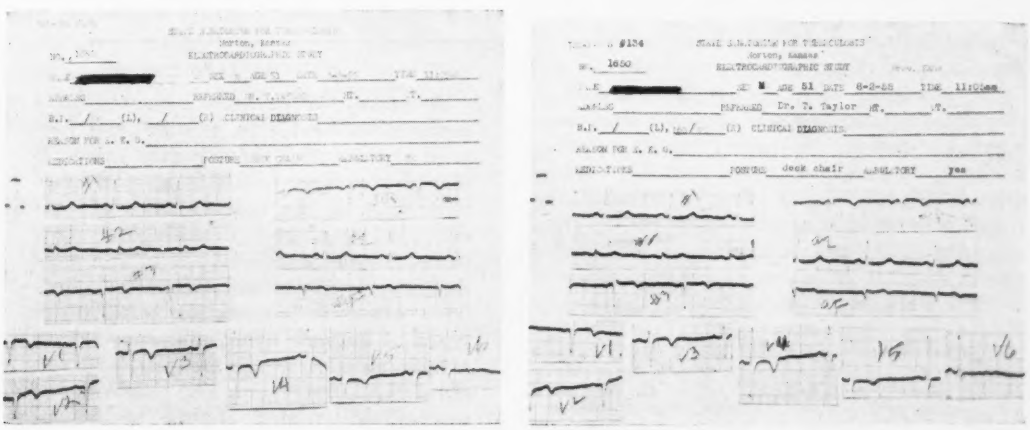

Figure 3. Comparison of the sending and receiving electrocardiogram. At left, sending; at right, receiving.

When several patients were studied in the course of one session, much valuable telephone time was initially lost in changing electrodes. We have since acquired another set of patient electrodes, and now one patient is being made ready while another is still connected to the sending apparatus. It is occasionally a problem to scan a complicated tracing quickly and report the diagnosis to the medical secretary on the sending end of the line. However, a review of our first 100 cases shows that errors picked up in a later deliberate study were of a minor nature. A cathode ray tube has been suggested as a guide in this scanning procedure[9] though we feel we can do as well with the direct writing tracings we now have.

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Intracranial Tumors

As Encountered in a Psychiatric Clinic

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Oddly enough, it is still a common belief among medical men that intracranial tumors belong in the category of rare and hopeless diseases. However, in modern neurology such tumors are regarded as both frequent and treatable. During the last 20 years the mortality rate from early surgery of these lesions has improved dramatically, even though about 50 per cent of the tumors treated by surgery are found to be malignant, either because of their nature or their localization. This is not discouraging to the neurosurgeon, however, as he can seldom be certain of the accuracy of his pre-operative diagnosis.

The importance of early diagnosis is obvious in view of the fact that growth of the tumor often causes irreversible damage. The classical triad of symptoms, headache, vomiting, and choked discs, is of limited value in diagnosis since it points to increased intracranial pressure rather than to tumor per se. Those symptoms usually indicate a history of longstanding growth or acute circulatory disturbance, either of which is a serious condition. Absence of the symptoms, on the other hand, does not exclude the possibility of intracranial tumor.

One is tempted to employ the generalization that early symptoms should be searched out. Unfortunately, general rules are of little value since symptomatology varies widely with the age, nature, and localization of the tumor.

Grinker[1] believes that "the only true symptom of an intracranial tumor is the gradual, progressive loss of neurological function." It is true, however, that

SUMMARY

A survey of patients admitted to a psychiatric service in a municipal hospital showed that unsuspected intracranial tumors were found in a small number. Most of the tumors were of considerable size, and neurological signs were frequently demonstrable. The most frequent differential diagnostic problems arose in connection with other types of organic brain diseases such as arteriosclerosis, brain atrophy, and cerebral vacular accident. The possibility of intracranial tumor should always be kept in mind in psychiatric patients.

Presented before a group of Topeka psychiatrists in 1952 from clinical material first reported by the author in *Nordisk Medicin*, published in Denmark. Dr. Wagner is now en route to Copenhagen, Denmark, where he will be a member of the staff of the Department of Psychiatry, Bispebjerg Hospital.

progressive functional loss occurs in a wide variety of neurological diseases and that tumors not infrequently give rise to acute or episodic incidents. Acute bleeding into a tumor may be confused with hypertensive encephalopathy, arteriosclerosis, and cerebral thrombosis. There may be an acute or subacute onset of symptoms following skull trauma which may be indistinguishable from that of subdural hematoma. Traumatic incidents are often noted in the clinical history of patients whose vision or coordination is impaired.

To make diagnosis more difficult, the optimum time for the appearance of tumors coincides nicely with the onset of many degenerative diseases. The unhappy clinician is therefore faced with the touchy and subtle problem of distinguishing arteriosclerosis, thrombosis, and so forth from neoplasm. Even the trained clinician may therefore find it difficult to decide how far to carry out his examination, especially with respect to air study.

While mental symptoms like drowsiness, stupor, or even coma may indicate severely increased intracranial tension, other symptoms such as intellectual impairment or gradual character change may be the initial findings long before the onset of any neurological signs. Frontal lobe tumors are especially capricious in that respect. The insidious onset of disinterest in environment, carelessness in personal appearance, blunting of emotional response, and diminishing attention span may remain unrecognized by close relatives for a long period of time. In later stages the extremely careless, incontinent, and untidy patient may be mistaken by the physician for a severely regressed schizophrenic. Psychological tests usually point to organic brain damage, but give no information about pathology, and seldom indicate localization unless the aphasic zone is involved.

The following material on intracranial tumors in a psychiatric hospital in Denmark with respect to type, localization, and symptomatology will compare the diagnoses made by the referring physician with those of the hospital staff and the pathologist. It should be remembered that statistics arising from a psychiatric hospital may not be comparable to those originating in other types of hospitals.

This review concerns patients in a 120-bed acute psychiatric admission unit in one of the municipal hospitals in Copenhagen.[2] It covers 12 years between 1939 and 1950, during which period 31,348 patients were admitted, an average of 2,612 patients per year. The diagnosis of brain tumor was established in only 49 cases, 18 men and 31 women, 0.15 per cent of the total admission. It is difficult to decide whether this low figure is due to lack of diagnostic skill on the part of the hospital staff or to the fact that most patients with brain tumors were admitted directly to neurosurgical wards. However, we omitted from the study patients who were reoperated upon, those who were readmitted, and those who had recently been operated on and transferred to the unit for psychiatric reasons.

TABLE I

	Front.	*Temp.*	*Par.*	*Hem.*	*Bas.*	*Mult.*	*Total*
Meningioma	7	3	1	0	2	0	13
Glioma	8	6	3	9	2	0	28
Metastases	3	0	0	0	0	5	8
Total	18	9	4	9	4	5	49

Table I shows distribution of the tumors as to type and localization. Gliomas represent about one-half of the cases, 28 out of 49, which is in accordance with general experience. Meningiomas, however, were slightly more frequent than the usually estimated 15 to 20 per cent, appearing in one-fourth of the cases. Most remarkable is the fact that one-third of all the tumors were localized in the frontal lobe, in contrast to the usually quoted 16 to 25 per cent. Eight of those 18 patients presented a typical lobotomy-like clinical picture. Only one was described as euphoric.

Among adults, intracranial tumors occur most frequently during the fourth to fifth decade of life. Table II shows that this study concerned a different age distribution, the maximum being in the fifth to the sixth decade. This holds true for meningiomas and gliomas as well as for metastases. Since one-fourth of the tumors occurred in the seventh decade, the differential diagnosis of organic brain disease in elderly people was a real problem.

Listing the symptoms the patients showed at the

TABLE II

			x
			x
			xx
			xx
			xx
			xxx
			xxx
			xxx
	x		xxx
	xx		xxx
x	xxx		xxxx
x	xxxx		xxxx
xx	xxxx		xxxx
xxx	xxxx	x	xxxx
xxx	xxxx	xxx	xxxxx
00xxx	xxxxxx	x0xxx0	xxxxxx
34567	345678	345678	345678
Meningiomas	Gliomas	Metastases	Total

time of admission would not indicate the specific diagnostic problems, but a survey of psychic and neurological symptoms will give an over-all impression as to their distribution. Four-fifths of the patients showed intellectual impairment, three-fifths had developed marked emotional changes, and one-half revealed impairment of consciousness varying from drowsiness to stupor. Neurological symptoms such as aphasia, epileptic seizures, and choked discs appeared with approximately the same frequency, in one-third of the patients. Hallucinations were found in only two patients where tumor involved the temporal lobe. It is especially important that more than three-fourths of the patients disclosed other neurological signs such as reflex abnormalities, paralysis of one of the extremities, or pathological changes in the spinal fluid.

Table III shows diagnoses by the referring physician, the hospital staff, and the pathologist. This gives a gross orientation as to the group of diseases in which the referring physician placed the patient. The table shows that about three-fourths of the tumors, 37 out of 49, were diagnosed clinically. Approximately half were verified at operation, the rest at autopsy. There were various reasons for the fact that not all patients with the diagnosis of intracranial tumor underwent surgery. Some were too old or in poor condition for surgery; a few died soon after admission. Autopsy examination of 20 of the 23 patients in whom tumor was diagnosed disclosed that there were 12 glioblastomas, 3 astrocytomas, and 5 metastases, mostly malignant tumors.

The tumor was diagnosed clinically on all the patients referred with the diagnosis of epilepsy, but only three-fourths came to surgery. This diagnostic efficiency may be due to the fact that the onset of epileptic seizures in adult life always causes suspicion of intracranial tumor and calls for a careful neurological examination and air study.

Patients admitted with a diagnosis of "senile dementia" constituted only a minor group numerically, but apparently diagnostic efficiency was especially low in this instance. As expected, the most frequent error was the confusion of tumor with cerebral arteriosclerosis or cerebral vascular accident.

TABLE III

Initial Diagnosis	*Staff Diagnosis*		*Verified*		
	Org. Brain Dis.	*Brain Tumor*	*Op.*	*Autopsy*	*Total*
Psychosis	8	11	10	9	19
Senility	3	2	2	3	5
C. V. A.	1	3	2	2	4
Epilepsy	0	16	11	5	16
Others	0	5	1	4	5
Total	12	37	26	23	49

An example is a 64-year-old woman who complained of gradual fatigue and drowsiness five weeks prior to admission. Two weeks before admission she developed speech difficulties and a severe memory defect. The examination on admission showed clouding of consciousness, marked motoric aphasia, left central facial paralysis, and hyperactive muscle reflexes on the same side. Fundi were normal. She died within a week, and autopsy showed a huge glioblastoma infiltrating most of the right hemisphere.

The following case history illustrates that a slowly progressive deterioration may be caused by a benign tumor of considerable size. A 69-year-old woman with a six-year history of anosmia and a two-year history of short periods of excitement and confusion became stubborn, sullen, and incontinent during the two months before admission. The admitting diagnosis was senility. She was a quiet, deteriorated patient who was sent to a neurosurgical ward because of a generalized convulsion which left her with a permanent Babinski reflex. An air study showed depression of the left ventricle, and a midline meningioma the size of a tennis ball was removed from the olfactory groove.

Presenile dementia may give rise to differential difficulties with brain atrophy. The following example shows the value of a thorough examination. A 49-year-old previously healthy man was transferred from a medical ward with the diagnosis of Alzheimer's disease. Four months prior to admission he had suffered from diarrhea, followed by temporary clouding of consciousness. Examination showed a deteriorated, hypokinetic, emotionally flattened patient with a left-sided Babinski reflex. Repeated pneumoencephalograms failed to show any filling of the ventricular system, a finding usually ascribed to technical error. On the chance that it might be caused by obstruction, however, the patient was transferred to a neurosurgical ward where a 130-gram meningioma arising from the anterior part of the superior longitudinal sinus was removed.

The greatest diagnostic problem was in the group of patients with non-organic psychiatric difficulties. Fourteen of 19 were labeled as having psychotic or neurotic depression. Half of the tumors in this group were diagnosed clinically, and in the remainder the symptoms were attributed to organic brain disease, usually a cerebral vascular accident.

This example of skull trauma is cited to show that misinterpretation may lead to an incorrect neurological diagnosis. A 52-year-old previously healthy woman was admitted to the medical ward because of

a post-traumatic headache. She had had a head injury three weeks before admission, and four days after the injury she developed headache on the right side. After three weeks on the medical ward she was transferred to the psychiatric service with the above mentioned diagnosis and a suggestion that she might be malingering. Examination showed a drowsy patient with a slight left-sided paralysis of the facial nerve and bilateral papilledema. A big glioblastoma was subsequently removed from the right temporal lobe.

In some instances organic symptoms may be preceded by a prolonged period of depression, likely to be accepted as such if precipitated by a psychic trauma, as in this example. A 51-year-old sensitive and melancholy woman continued to be severely depressed for five years following the death of her husband. Two months before admission she became apathetic and drowsy and mentioned several times that she thought she was going to die. On admission she presented the picture of a lobotomized patient, friendly, somewhat silly, indifferent, and having a short attention span. She soon became disoriented and hyperactive, voided in bed, and smeared herself with feces. Positive neurological findings were slight protrusion of the right eyeball and a bilateral Babinski sign. The neurosurgical diagnosis was frontal lobe tumor, and operation revealed a big anterior parasagittal meningioma.

Information about the pre-morbid personality is of importance in evaluating character changes caused by a developing tumor. An aggravation of a neurotic condition, during the menopause for instance, may be misleading if specific points in the clinical history do not lead one aright.

A 48-year-old sensitive, exaggerating woman was admitted with the diagnosis of migraine and neurotic depression in the menopause. During the four months before admission she had suffered from paroxysmal left-sided headache, nausea, and vomiting. She had occasionally had difficulty in finding the correct words to express herself. She was described on admission as friendly, indifferent, and without remarkable depressive features. Neurological examination was negative. During the next week she became apathetic, and during the second week she developed a slight amnestic aphasia, right-sided paralysis of the facial nerve, and hyperactive muscle reflexes on the same side. Since a tumor in the left frontotemporal region was suspected, she was transferred to a neurosurgical service, and a meningioma the size of a baseball was removed from the left sylvian fissure, extending from the sphenoidal ridge.

Deep-seated gliomas frequently give cause to an acute psychotic condition, often showing the first neurological symptoms in the terminal stage, as in the following instance. A 50-year-old previously healthy woman was admitted with the diagnosis of menopausal psychosis. Twelve days before she had been dismissed from surgical service where she had received treatment for a tibial ulcer. During her hospitalization she went through a short period of elation, but at the time of her discharge she was described as being in a normal mental condition. Immediately after leaving the hospital she became anxious, depressed, and sleepless, vomited, and complained about paresthesia in both hands. On admission to the psychiatric service she was disoriented in all three areas, was aphasic, and talked all the time. During the following week she developed paralysis of the right arm, became incontinent and comatose, and died suddenly. Autopsy revealed a cystic astrocytoma in the left parietal lobe, about the size of an orange and expanding into the lateral ventricle.

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Imagination is potentially infinite. Though actually we are limited to the types of experience for which we possess organs, those organs are somewhat plastic. Opportunity will change their scope and even their center. . . . The precision and variety of alien things fascinate the traveler. He is aware that however much he may have seen, more and greater things remain to be explored, at least ideally, and he need never cease traveling, if he has a critical mind. —*George Santayana*

The Breast

A Review of Its Inflammatory Lesions and Tumors

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SUMMARY

A review of the embryology and anatomy of the breast and its various inflammatory lesions and tumors.

EMBRYOLOGY

Mammary glands first appear during the sixth week of human development as an ectodermal thickening called the milk line. Human mammary glands subsequently develop from this thickening as down growths of solid cords, which have been called the primary milk ducts. These later branch in the corium, hollow out, and, at their ends, dilate into secretory acini. Where the "milk ducts" open on the surface, the epidermis is elevated to form the nipple.

The mammary glands are regarded by most authorities as modified sweat glands, because of their similar mode of development and structural arrangements. Rudimentary mammary glands (areolar glands of Montgomery), which also resemble sweat glands, occur in the area about the nipple. By the time of birth little more than the main ducts are present, with their terminal acini projecting down into subcutaneous tissue. These remain as such until puberty, when the areolar region becomes elevated and a rapid enlargement takes place through fat deposition about the glands.

There will be more about this later when we discuss malignant tumors of the breast.

ANATOMY

The glandular tissue forms 15 to 20 lobes arranged radially about the nipple, each lobe having its own individual excretory duct which has an ampullary enlargement just beneath the areola. The glandular tissue does not occupy the entire eminence called the breast; a variable but considerable amount of adipose tissue fills out the stroma between and around the lobes. The connective tissue stroma in many places is concentrated into fibrous bands which course vertically through the substance of the breast, attaching the deep layer of the superficial fascia to the corium of the skin. These bands are known as the suspensory ligaments or Cooper's ligaments of the breast.

Minute processes of glandular tissue may accompany these fibrous bands into the retro-mammary tissue and thence, via septa of the pectoral fascia, between the bundles of fibers of the pectoralis major muscle. Malignant growth of the breasts may extend along these septa, a fact to be remembered in surgical removal of the breast.

The entire breast is contained within the superficial fascia. The deep surface of the gland is separated from the underlying external investing layer of deep fascia by a fascial cleft which allows considerable mobility. Each lobe of the breast consists of numerous lobules, connected by areolar tissue, blood vessels, and ducts. The lobules consist of a cluster of rounded alveoli which open into small lactiferous ducts. These ducts unite to form larger ducts and subsequently end in a single canal for each lobule. These ducts are void of muscle tissue but contain elastic fibers. They are lined with columnar epithelium, resting on a basement membrane.

The arteries supplying the breasts are derived from three main sources. These are the intercostals, internal mammaries, and the axillary arteries with their thoracic branches. The venous drainage system begins with an anastomotic circle around the base of the papillae of the ducts, beneath the areola. From this, large branches pass to the circumference of the gland and end in the axillary and internal mammary veins.

The nerve supply is laterally from the lateral mammary rami of the anterior rami of the lateral cutaneous branches of the fourth to the sixth intercostal nerves and medially from the medial mammary rami of the anterior cutaneous branches of the second to the fifth intercostal nerves. In addition the skin receives branches from the supraclavicular nerves of the cervical plexus. Therefore, in painful affections of the breast, the pain may be referred to the side of the chest and back, along the intercostal nerve trunks, over the scapula, along the medial side of the arm, along the intercostal brachial nerve, or up into the neck. Sympathetic fibers reach the gland, but by what course is not yet clear.

The lymphatics of the breast are numerous. There is a rich plexus in the skin of the areola and nipple which empties mainly into a subareolar plexus. Deep lymphatics arise in the spaces around the alveoli in all parts of the gland. They anastomose freely with the cutaneous lymphatics, and most of them converge toward the nipple where they join the subareolar plexuses.

From these the main lymphatics course outward along eight principal lymphatic pathways:[1] (1) axillary route, to anterior pectoral nodes (low), cen-

tral axillary nodes (mid), to subclavian nodes (high or apex); (2) internal mammary route along the internal mammary artery to mediastinal nodes; (3) paramammary route of Gerota, through the abdominal lymphatics to the subdiaphragmatic nodes; (4) Groszman's path from lymphatics beneath the breast perforating the pectoral major muscle to Rotter's nodes, thence to subclavian nodes; (5) cross mammary pathway via superficial lymphatics to the opposite breast; (6) substernal pathway to the mediastinal nodes; (7) subclavian pathway direct to the subclavian nodes, and (8) lower superficial pathway to the lymphatics of the abdominal network.

DIAGNOSIS

With this knowledge of the development and anatomy of the breast, let us proceed further with the problem at hand, diagnosing an existing pathological condition of a breast. History, inspection, transillumination, palpation, roentgenograms, and biopsy of the lesion are all used to reach this end. However, microscopic study is the only reliable basis on which to establish the final diagnosis in mammary lesions.

Clinically, if one takes into consideration the age of the patient, whether the lesion is single, multiple, or diffuse, the location of the lesion, its characteristics on palpation, the presence or absence of inflammatory signs and evidence of injury, the number of possibilities to be considered in the differential diagnoses is definitely limited.

Injury may be of significance in two mammary lesions: hematoma and traumatic fatty necrosis. Injury of the nipple during nursing may be a factor in lactation mastitis. Inflammatory signs are present in acute and chronic lactation mastitis and in some cases of tuberculosis and syphilis of the breast. Such signs are found in cases of dilated ducts beneath the nipple with an associated plasma-cell mastitis. An erysipeloid appearance of the skin is seen in cases of acute cancer, and reddening and edema with or without ulceration and infection are seen in the late stages of all forms of mammary cancer.

The age of the patient is important in determining the physiologic state of the mammary gland. The decade between 15 and 25 years is the period of adolescence and early sexual maturity. This is the time of appearance for most cases of diffuse virginal hypertrophy and benign fibroadenoma. The decade between 30 and 40 years in nonparous women is the period of menstrual cycles, and it is at this time that the various forms of mammary dysplasia or cystic mastitis are found.

In relation to childbearing, naturally, are found acute and chronic forms of lactation mastitis, galactorrhea, and galactocele, and the rapidly growing fibroadenomas and cancers of pregnancy.

At the time of the menopause large fibroadenomas or giant myxomas may appear, also dilated ducts beneath the nipple with inspissated secretion, papillomas beneath the nipple associated with a sanguineous discharge, solitary cysts, and the various forms of mammary cancer. With advancing age, mammary carcinomas and fibrosarcomas of the breast are the predominant lesions.

Most mammary lesions are solitary rather than multiple, and few are bilateral or diffuse. Mammary hypertrophy in the male and female is diffuse and often bilateral. Among the types of mammary dysplasia or cystic mastitis, adenosis is most often a multiple and bilateral condition. Cystic disease is multiple and bilateral in some instances, and small intracystic papillomas may occur in multiple form and may be bilateral. Dilated ducts with inspissated secretion are often multiple and may be bilateral. Recurrent mammary cancer may give rise to multiple nodules (so-called carcinoma en cuirasse). Large fibroadenomas during adolescence, giant myxomas, and large mammary cancers and sarcomas may occupy the entire breast and give the impression of a diffuse lesion.

Most mammary lesions are found in the glandular tissue in any of the various portions of the breast. A few have a characteristic location beneath the nipple. Such lesions are papillomas in the larger ducts, dilated ducts beneath the nipple, Paget's cancer of the nipple, and some forms of papillary cancer and duct cancer.

The most important data from the standpoint of clinical diagnosis are those obtained by palpation. Whether the lesion is soft or circumscribed, firm or infiltrating, is important. Cysts, benign papillomas, dilated ducts, and the papillary and mucoid forms of mammary cancer may be soft or circumscribed on palpation. The same is true of mammary abscesses, galactoceles, lipomas, and the rare angiomas. The tender tissue in mastodynia, benign fibroadenomas, and nonsuppurating mastitis is firm but not infiltrating. The common forms of mammary cancer are hard, infiltrating lesions. This is also true of fat necrosis and most forms of sarcoma.

Geschickter,[2] in his book *Diseases of the Breast*, gives a list of 12 "don'ts" which I think is worth repeating.

1. Do not overlook early pregnancy in taking menstrual and marital history; this may be responsible for the sudden growth of a benign or malignant tumor.

2. Do not neglect a history of puerperal mastitis. Cancer occurring in the scar of lactation mastitis has a grave prognosis.

3. Do not neglect microscopic examination of any discharge from the nipple. This is necessary to distinguish between blood and inspissated secretion.

4. Do not neglect transillumination. It may reveal a nonpalpable tumor.

5. Do not palpate the breast with the patient in the upright position, but have her lie flat and palpate the gland against the chest wall with the flat of the palm or the tip of the fingers, using gentle pressure. Include the region of the axillary and supraclavicular nodes in the palpation.

6. Do not fail to determine the mobility of the tumor, if present, the mobility of the overlying skin, and that of the nipple. Evidence of traction or adherence of any of these structures may be the earliest sign of malignancy.

7. Do not omit a pelvic examination. It is important in the diagnosis and treatment of chronic cystic mastitis.

8. Do not omit a Wassermann reaction. A lesion of the nipple cannot be diagnosed without a serologic test.

9. Do not rule out the possibility of cancer without a biopsy if a definite nodule can be palpated or transilluminated.

10. Do not do a biopsy without being prepared to interpret the section and to proceed with a radical operation if indicated.

11. Do not omit roentgenograms of the chest and pelvis (including the lumbar spine) if a radical mastectomy is contemplated.

12. Do not give endocrine or radiation therapy unless the diagnosis is established.

DISEASE ENTITIES

In discussing the various disease entities of the breast, I wish to take up only a selected few of the less common ones and to go into greater detail about the group classified as chronic mastitis and tumors, both benign and malignant.

Fat Necrosis: Destruction of fat followed by regenerative process in which phagocytoses and foreign body giant cells are prominent may occur in the breast with a variety of conditions such as abscess, benign and malignant tumors, and following trauma, and may be confused clinically with carcinoma. This usually appears as a small, dense nodule and may show fixation to the skin and induration suggesting cancer. Grossly, the affected area appears encapsulated and without the infiltration seen with malignancies. On section some of these will be discovered to contain cystic cavities with cholesterol crystals and calcinous deposits. These give the lesion its hardness to palpation. The fibrous repair easily explains the skin retraction as it acts like a strong cord which is nonelastic and tends to retract. On microscopic sections, these lesions appear as granulomatous processes in which fibrous tissue, lymphocytes, and giant cells occur together with the characteristic foam cells which are lipoid phagocytes.

Plasma Cell Mastitis: This rare condition may easily be mistaken for carcinoma, but its rapid onset, local tenderness, and puriform discharge from the nipple, together with mild temperature elevation and leukocytosis usually suffice to make the diagnosis of plasma cell mastitis. Adair[8] described this as periductal mastitis arising in association with dilated ducts beneath the nipple. Because of the induration and edema of the skin, retraction of the nipple may be present. Usually the findings increase in severity over a considerable period of time, to be followed by slow resolution, leaving an indurated nodule. One of the most characteristic findings is the presence of one or more tense bands or cords, which are distended ducts, traversing the inflamed areas and from which a thick, dark discharge may be expressed on pressure. The microscopic picture is one of subacute inflammation with plasma cells predominating. Giant cells may be numerous also.

Chronic Cystic Mastitis: We now approach a subject which is the root and center of many of the difficulties in diseases of the breast. The first problem is to decide by which name the condition should be known. Its names are indeed legion. The better known fall into three groups: (1) chronic mastitis, chronic cystic mastitis, chronic interstitial mastitis, (2) diffuse fibroadenoma, cystadenoma papilliferum; (3) involution cysts, abnormal involution, cystic disease of the breast, and Schimmelbusch's disease.

It is not that the name itself is of great importance, but almost all of these names commit one to a definite theory. The first group suggests that the condition is inflammatory in nature, the second that it is neoplastic, and the third that it is a perversion of involution.

The only real evidence in support of the inflammatory hypothesis is the presence of lymphocytic infiltration, and that is always found in involution following lactation and often in senile involution.

Nor can we admit that the condition is a neoplasm. There is no tumor—that is a fact from which we cannot escape. It is true that the epithelial hyperplasia may at times suggest a neoplastic process, but the same may be said of the epithelial hyperplasia in goiter. A reasonable explanation, and one which brings the condition into line with similar conditions in the thyroid and the prostate, is arrived at by comparing the account of the physiological changes which the breast undergoes throughout life with that of the lesions in chronic mastitis.

The breast is being continually played on by a variety of stimuli which tend to induce hyperplastic changes followed later by involution. We shall find that all the changes characteristic of hyperplasia and of involution may be duplicated in chronic mastitis (lobular hyperplasia). There is evidence of epithelial activity and proliferation in the form of epithelial

buds, later atrophy of the epithelium, cyst formation, fibrosis, and round cell infiltration—in short, evidence of hyperplasia which may or may not be associated with involution.

The patient complains of pain or a lump in the breast. The pain, although usually slight, may be severe, is often worse at the menstrual period, and frequently is neuralgic in nature. The breast is tender, especially at certain points. One or both breasts may be involved, and there may be more than one lump in one breast. This multiplicity is always strong evidence against cancer.

A granular induration is felt when the breast is palpated between the fingers and thumb, much less distinctly when the flat of the hand is used. In this respect, it differs from both innocent and malignant tumors. In thin persons the breast substance has a characteristic ropy feeling. Cysts feel hard rather than fluctuating and give the induration a more coarsely granular character. The induration is frequently confined to one of the sector-like lobes of the breast, whereas in cancer no such restriction is observed. The axillary nodes are often enlarged and tender. As in carcinoma, they may not be palpable. The condition of the nodes should not be used as a means of diagnosis. The cause of the glandular enlargement in lobular hyperplasia is unknown. Although the disease is most common at the time of menopause, it may occur before the 13th year. Bloodgood[4] states that it is more frequent in the breast which has never lactated, whether there has been pregnancy or not.

By far the most common type of cyst is what Bloodgood calls the blue-domed cyst. Its surface is smooth and the fluid contents are clear or cloudy, never hemorrhagic. Hemorrhage into a cyst indicates carcinoma.

Microscopically one sees an extremely varied picture. Considered individually, the different features are largely meaningless, but when regarded as parts of one process of epithelial hyperplasia or retrogression they become more intelligible.

The principal changes are as follows: (1) A formation of epithelial buds is similar to those seen during the involution of the breast at puberty; (2) Cyst formation is extremely common (Cheatle[5] believes that the majority of cysts are formed not from the acini but from the ducts); (3) Papillary formation is a characteristic change in many cases; (4) The presence of large, clear, pale, epithelial cells is sometimes noted; (5) Atrophy of the epithelium rather than hyperplasia may be the dominant factor, and (6) The connective tissue usually shows a significant increase. This is best observed in the periductal fibrous tissue.

While we are discussing this common disease entity, let us ask the question, "What is the relationship of this lobular hyperplasia to carcinoma?" The modern tendency is to regard cystic mastitis as a dangerous and precancerous condition. We must remember that this hyperplasia is the result of hormonal stimulation and that, at least in the experimental animal, carcinoma of the breast can be induced by means of estrogenic hormones.

Shields Warren[6] gives the follow-up results after five years on 1,200 patients with chronic mastitis who had been operated upon and compares them as regards cancer incidence with a control group of corresponding age. He finds that the cancer rate for women with pre-existing breast lesions is 4.5 times as great as in controls; between the ages of 30 and 49 it is nearly 12 times as great. Warren concludes that a woman who has had chronic mastitis is in far greater danger of developing cancer, even though all the apparently abnormal tissue has been removed, but once she has passed the menopause there is no greater danger than in any control group.

Actively proliferating lesions, such as intraduct papillomas and cysts with papillary epithelium, are of graver import than large cysts with atrophic epithelium. After removal of the diseased tissue, the breasts should be examined at regular intervals. This is better than amputation of both breasts, which is not only too radical but too easy for the surgeon. Much, however, depends on the type of lesion and the age of the patient.

Schimmelbusch's Disease (Adenosis): Adenosis is characterized by the occurrence, in one or both breasts, of multiple nodules varying from a millimeter to a centimeter in size, usually distributed about the periphery of the upper or outer hemisphere. The breasts affected are small, dense, and edged like a saucer when grasped in the hand. Pain and tenderness (which vary during the menstrual cycle) occur as in mastodynia. The majority of women affected are childless and are in the late 30's or early 40's. These patients are often nervous and underweight and may have irregular menstrual cycles.

The mammary tissue affected contains dense fibrous tissue, numerous minute cysts, and foci of epithelial proliferation. The pathology of this form of mammary dysplasia was first described as a diffuse papillary cystadenoma by Schimmelbusch, who believed it to be precancerous. Bloodgood, in 1906, described it as the "diffuse nonencapsulated cystic adenomatous type" of chronic cystic mastitis.

The occurrence of small multiple nodules has been emphasized. These numerous small nodules found in the upper or outer hemisphere of the mammary gland have led to descriptive terms such as "shotty breasts," "the feeling of a bag of shot" and "palpating a bean-bag." The size of the nodules varies from 1 or 2 mm. to 1 or 1.5 cm. The larger nodules are cystic, being round, dense, and freely movable. In the ma-

jority of cases of adenosis, both breasts are affected. Bloodgood[7] believed that if both breasts are carefully palpated, bilateral disease would always be found.

Pathologically, there is a diminution of adipose tissue and increase of fibrous tissue. The parenchyma is riddled with small cysts, minute adenomas, papillomas, and large dilated ducts. Microscopically,[8] the salient features are (1) epithelial proliferation in the terminal mammary tubules with the formation of multiple small intraductal adenomas and papillomas —intraductal hyperplasia; (2) a disorderly proliferation of acinar elements which invade the surrounding stroma, so-called "epithelial spilling"; (3) dilatation of terminal tubules or acini with the formation of minute cysts—microcystic disease—and (4) increase in the periductal and perilobular stroma—diffuse fibrosis.

Tumors of the Breast (Benign): Of the true tumors of the breast, 95 per cent fall into two groups, fibroadenoma and carcinoma. The former constitutes about 15 per cent, the latter 80 per cent. The fibroepithelial breast tumors are essentially fibroepithelial overgrowths which are limited by a capsule. Sometimes the fibrous element is predominant, sometimes the epithelial. The development of cysts will materially modify the character of the tumor.

Three main varieties of fibroepithelial tumors may be recognized: (1) pericanalicular fibroadenoma; (2) intra-canalicular fibroadenoma, and (3) duct papilloma. Clinically, these types can't be differentiated.

In 600 cases of fibroadenoma reported by Geschickter, the age incidence was highest between 21 and 25 years. Eight per cent occurred in women past the menopause, and in about 10 per cent it occurred before the first menstruation. These tumors usually appear as a solitary lesion and most often in the upper, outer quadrant of the breast. The breasts are usually well developed, firm, and of the virginal type.

Microscopically, these tumors can be differentiated. The pericanalicular fibroadenoma appears as an overgrowth of both fibrous and epithelial structures. The acini and the ducts appear more numerous than normal and are surrounded by a stroma of fibrous tissue which varies greatly in density, and which is most evident around the ducts, accounting for the name periductal or pericanalicular. Although resembling the structure of the normal breast, that structure is never perfectly reproduced. The glandular arrangement is more diffuse, and new lobules are not formed.

The *intracanalicular type* of fibroadenoma is more a fibroma than an adenoma. It has a remarkable proliferation of connective tissue which projects into the ducts in the form of polypoid masses, producing great elongation and distortion of the ducts. The ducts are usually much dilated. On cut section the dilated ducts appear to be crowded with masses of fibrous tissue, each covered with a layer of epithelium invaginated from the duct wall and separated from one another by a series of branching slits.

The third type of fibroepithelial benign growths to be considered is the *duct papilloma.* Although there is a superficial resemblance between duct papilloma and intracanalicular fibroadenoma, there is one essential difference; in the former the actively growing element is epithelium, while in the latter it is fibrous tissue. The duct papilloma also appears to have a definite relationship with duct carcinoma.

Grossly, this tumor appears as a little papillomatous growth which projects into a dilated duct, usually close to the nipple. Microscopically the picture is again a varying one. The early stage will show a typical papillary or villous formation, the delicate stroma being covered by a single layer of columnar epithelium. The larger specimens appear to have an adenomatous structure because of gland-like spaces formed by the interlacing processes. It is on this account that such misleading terms as adenocystoma and cystic adenoma have been applied to the condition. The blood vessels are large and thin-walled so that hemorrhage is common, and a discharge from the nipple, either blood-stained or serous, is one of the most characteristic symptoms.

We might also include under this classification of fibroepithelial tumors of the breast one first described by Johannes Muller[9] as a giant mammary myxoma and called "cystosarcoma phylloides." These often grow to immense sizes and occur near the menopause. There are none of the familiar skin changes that appear with carcinoma of the breast. These large tumors have cystic spaces into which fibrous polypoid masses project. Microscopically, myxomatous connective tissue predominates, and the polypoid fibrous structures are covered by layers of cylindrical or cuboidal epithelium.

Malignant Tumors of the Breast: Let us now turn to malignant tumors of the breast. A universal classification of such is wanting, but if we look at mammary cancers as of two fundamental pathological types we can arrive at a workable classification. These are (1) the adenocarcinomas, arising from lobular structures and growing either in an infiltration or circumscribed fashion, and (2) the stratified epithelial cancers, derived from tissues concerned in the development of the nipple and larger ducts.

The adenocarcinomas or gland-cell cancers comprise 90 per cent of cancers of the breast. The gland-forming tendency of the infiltrating form (often referred to as scirrhous cancer) is not apparent under the microscope except in the more slowly growing tumors. The less common, circumscribed adenocarcinomas, comedo, papillary, and gelatinous, which grow more slowly, have more definite glandular ar-

rangements, and this group is usually referred to as adenocarcinoma.

Stratified epithelial cancers are derived from epithelium, resembling that found in the normal epidermis, and include Paget's cancer of the nipple, large cell or pagetoid duct cancer, so-called medullary cancer, and cancer cysts.

Rare forms of cancer not belonging to these two major groups include squamous cell cancer, usually complicating benign epidermoid cysts, adenocystic basal cell cancer, and cancers derived from the sweat glands.

GLAND CELL CANCERS

Infiltrating Adenocarcinomas: Approximately 75 per cent of all mammary carcinomas are of this type. These originate in the small tubules or acini which make up the lobular structure. Since the acini arise by finer ramification from the terminal tubules, there is no definite line between the two. The terms carcinoma simplex, scirrhous carcinoma, and lobular cancer are also applied to this disease process. These tend to rapidly invade the stroma.

Infiltrating carcinomas of the breast are relatively rare under the age of 26 years; however, they may occur any time after the onset of mammary development. Seventy-five per cent of all cases occur between the ages of 35 and 60 years, and 50 per cent occur between 40 and 55 years.

An examiner may have difficulty in differentiating infiltrating carcinoma of the breast from fibroadenoma, a deep-lying, thick walled cyst, residual lactation mastitis or plasma-cell mastitis, fat necroses, and indurated tissue in adenoses. Microscopically, the cells are seen to grow characteristically in small nests or narrow elongated cords with intervening fibrous tissue and, rarely, a small amount of lymphoid infiltration.

Comedo Carcinoma: Current discussions about carcinoma of the breast rarely consider the less common circumscribed forms such as comedo, papillary, and mucoid or gelatinous adenocarcinoma which differ greatly in their manner of growth and prognosis from the infiltrating form just described. Comedo carcinomas were first described by Bloodgood as slowly growing cancers which arise from the terminal ducts. These have a tendency to invade preexisting ducts and to be confined by the walls of the ducts.

Because comedo carcinoma arises within the terminal tubules which have lost most of the lobule-forming capacity that is seen in infiltrating carcinoma of the breast, and since vascularity in this area is less, these growths metastasize relatively late and have in general a favorable prognosis. It is significant, therefore, that no case of adenocarcinoma of the comedo type has been described in connection with pregnancy. It is seen in women past the menopause, or in younger women with a history of surgical castration, sterility, or of chronic cystic mastitis.

The cut surface looks like the cross section of a freshly sawed plank of wood. On pressure, plugs of tumor cells may be expressed from the ducts like comedos from a blackhead. Under the microscope, rings of carcinoma cells line the pre-existing and newly formed ducts, and small acinar spaces form within the thickened rings of cancer cells. Rarely, this may assume the role of an infiltrating carcinoma, as seen when the carcinoma cells infiltrate beyond the basement membrane of the ducts and invade the fat and fibrous tissue. This, of course, adds materially to the gravity of the prognosis.

Papillary Adenocarcinoma: The largest group of circumscribed mammary carcinomas shows a papillary or adenocystic structure. They may occur at any time after onset of menstruation. They are usually of large, bulky size, situated centrally beneath the nipple, and about 14 per cent are associated with discharge from the nipple. They may arise from preexisting benign intra-cystic papillomas. These tumors, like the infiltrating adenocarcinomas, also arise from epithelium destined to form mammary lobules. They tend to grow in coils and papillary folds and form acinar structures. The epithelium from which they arise, however, is not usually of the functioning variety. Instead, it represents remnants of the primary mammary sprouts from which the lobular tissue eventually forms, but which have been left behind in the region of the larger ducts in the nipple zone. These tumors are usually found free in the dilated ducts, attached only by a stalk. It is at this point that invasion into the stroma occurs. Pathologically, these tumors have a papillary and cystic structure and may contain necrotic tissue or hemorrhagic fluid.

Because of the vascularity of these growths and pedicle-like arrangement of their nutrient vessels, these are among the most radiosensitive of the mammary cancers.

GELATINOUS OR MUCOID ADENOCARCINOMA

Mucoid degeneration occurs in some slowly growing papillary adenocarcinomas. Whether this mucoid or gelatinous substance is secreted by the epithelium or is a degenerative change in the connective tissue, or whether both may occur, has been the subject of controversy. An epithelial origin of the gel is most probable, according to Broders.

These gelatinous growths have individual peculiarities. They usually grow slowly and metastasize late; some of the largest mammary cancers described have been of this character. Mucoid change may affect any of the glandular forms; it is most often seen, however, as a degenerative change in papillary adenocarcinoma. Mucoid cancers are soft and may have a cystic or fluctuant feeling on palpation.

When cut across, a semi-fluid, tapioca-like material oozes or flows from the incision. Under the microscope the nests of epithelium are widely separated by the pale-staining gelatinous substance. Recurrence due to transplantation of this material into the wound may take place years after operation.

STRATIFIED EPITHELIAL CANCERS

In the development of the breast, the nipple bud, forming from a downgrowth of epidermis, precedes the formation of the mammary sprouts from which the lactiferous ducts, the terminal tubules, and the lobular structures are derived. The invaginating epithelium of the nipple bud undergoes hyperdifferentiation and the central squamous cells are desquamated, forming the hollow of the nipple pouch. Prolongations of the stratified epithelium from this pouch line the openings of the larger mammary ducts. Cancers forming from this epithelium have characteristically large cells with granular, eosin-staining cytoplasm and have been grouped together under the heading of statified epithelial cancers.

Paget's Disease: This comprises 5 per cent of mammary cancers and is characterized by lesions of the nipple and by presence of large Paget's cells in the epidermal coverings of the nipple and with few exceptions in the mouths of the adjoining ducts. Broders[11] feels that this is a picture of carcinoma in situ. He also states that in 99 per cent there is an associated deep lying adenocarcinoma and that in the other 1 per cent it is there too but has not been found. Some authors maintain that the large granular Paget's cells are of sweat gland origin. This resemblance to cells of the apocrine sweat glands is understood when it is recalled, from an evolutionary standpoint, that the breast is a modified sweat gland and that these epidermoid-like cancers arise from relatively primitive mammary structures derived from the nipple pouch.

Intra-Duct Carcinoma: This group is less frequently reported than the Paget's type and is one in which the cancer affects chiefly the mammary ducts. Histologically the tumor tissue is identical with that formed in Paget's cancer of the nipple, but, as a rule, younger patients are affected and the clinical course is more rapid and malignant.

Medullary Cancers: This is another group of carcinomas which arise from transitional epithelium rather than from mammary glandular epithelium. In these, the tumor arises deep in the substances of the breast, and neither the nipple nor the large ducts adjacent to it are primarily affected. These may grow to a large size and undergo cystic degeneration, or they may remain relatively circumscribed and have a grandular structure, resembling sweat gland cancers. Whether they arise from derivatives of the primitive nipple pouch or owe their microscopic peculiarities to a process of metaplasis is difficult to determine.

NEOMAMMARY CANCERS

For the sake of completeness let me merely mention a group of neomammary cancers, to include intramammary stratified epithelial cancers, sweat gland cancers and circumscribed epidermoid cancers. Stratified epithelial cancers may occur deep in the breast and resemble histologically either nonkeratinizing epidermoid carcinoma of the skin or cancer arising in sweat glands. While in most instances either epidermoid or glandular features predominate, in some instances a combination of both is found. When it is realized that the breast is a modified sweat gland derived from the epidermis, it is easy to comprehend why these tumors originating in archaic epithelium resemble the epidermis in their undifferentiated portions and the sweat glands in the more highly differentiated parts. Despite their highly malignant appearance under the microscope, these cancers in which glandular features are prominent have a relatively good prognosis even when of large size and when they occur in women younger than 40 years.

MAMMARY SARCOMAS

Because of the rarity of these tumors, I want to mention them without further discussion. If the epithelial and mesenchymal elements of the breast were equally susceptible to malignancy, sarcoma rather than carcinoma would be the most frequent, since the latter exceeds the former in amount except during the periods of late pregnancy and lactation.

While the total number of sarcomas of the breast is small, the pathologic variety is great. Those originating in the fibrous stroma of the gland are varieties of fibrospindle-cell sarcoma which grow to immense size and metastasize relatively late. They are of two forms: (1) mammary fibrosarcoma which is primary in the connective tissue, and (2) mammary adenosarcoma which is secondary to fibroadenoma and contains scattered epithelial elements. Approximately one-half of all the sarcomas in the mammary gland are of these two varieties. Of the non-indigenous sarcomas of the breast, there are the osteogenic sarcomas, lipo- and myosarcomas, lympho-, myelo-, and angiosarcomas, and sarcomas of nerve sheaths and melanosarcomas.

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THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

The second session of the 84th Congress is under way, and in medical legislation—as in all other fields—this promises to be much livelier than last year's deliberations.

For one thing, neither the Republican administration nor the Democratic party, which is in control on Capitol Hill, got anywhere near as much as it wanted last year in medical legislation.

For another thing, and something that shouldn't be lost sight of at any time, both parties this year will be legislating with one eye cocked toward next November, when the voters make a choice between the two parties. Try as they might to pass laws for the good of all the people, neither party can afford to ignore the political realities of the situation: each will want to take credit for any legislation with popular appeal or, where that is impossible, at least to see that the other party doesn't get the credit.

In front of this political mosaic, these are some of the medically-important issues that will be fought out in Senate and House:

1. Federal guarantee of mortgages on health facilities. This has been on the Congressional calendar for two years; it was pushed hard in 1954, and was given some consideration in 1955. It would mean that the federal government would underwrite mortgages for hospitals, clinics, and nursing homes, under certain conditions, thereby allowing some sponsors to obtain loans they couldn't otherwise get, or to obtain them on longer terms and with lower interest.

2. Federal grants for research facilities. Under this plan—approved last session by the Senate—the U. S. would make outright grants to laboratories, medical schools, and clinics for building facilities for research in specific diseases, such as cancer and heart disease.

3. Federal aid to medical education. This perennial project probably is closer to Congressional enactment now than ever before. The most popular bill is one restricting the federal role to grants for building and equipment, with a financial incentive held out to those schools willing to increase their enrollment. This bill may be tied in with some other grants bill, such as the one for research.

4. Salk vaccine. Legislation authorizing federal appropriations for the purchase of Salk poliomyelitis vaccine ($30 million for the current year) expires February 15, virtually insuring Congressional action of some sort before that date. One issue is whether the federal government should continue the grants; more controversial is the question of whether the U. S. should move in to control the allocation and distribution of the vaccine. Allocation and distribution now are handled under a voluntary program supervised by the U. S. Public Health Service.

5. Increases in federal appropriations for medical research. Over the last few years—since the National Institutes of health came of age—Congress repeatedly has increased research grants over the amounts the Budget Bureau allowed Public Health Service to request. Indications are that this year the Budget Bureau may have to give way and allow important increases to be requested of Congress. Congress probably would want to add on its own special additions anyway, resulting in more money than ever before available for work on cancer, heart disease, mental illness, arthritis, blindness, and the many other conditions.

6. OASI-covered persons could receive payments beginning at age 50 if determined to be disabled. Under present law retirement payments for all are available at age 65. The bill containing this provision (H. R. 7225) passed the House last session by an overwhelming margin. It is now before the Senate Finance Committee, where the next phase of the legislative contest will be fought out in 1956.

The lop-sided House vote on disability payments may be discounted in part because of the parliamentary maneuvering by sponsors of the legislation. House members had only 40 minutes to debate this bill, and no opportunity to amend it. It was a case of accepting the whole bill—which contains a number of other social security liberalizations not of medical significance—or being politically damned as opposed to social security per se.

The American Medical Association maintains that the present expanding rehabilitation programs would be undermined by cash payments for disability, that the financial and other long-range aspects of the disability payments plan have not been thoroughly studied, and that the machinery for disability payments would inevitably project the federal government deeply into the medical care picture.

PRESIDENT'S PAGE

DEAR DOCTOR:

Happy New Year!

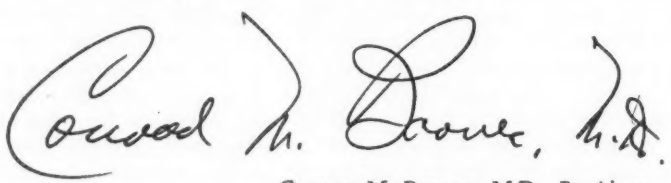
Thank you for the extension of Christmas greetings through your A.M.E.F. contributions. And, speaking of contributions, the John Porter Memorial Fund now stands at $4,245.

The A.M.A. meeting in Boston was both interesting and instructive to me. The lectures, scientific exhibits, and deliberations of the House of Delegates were all smoothly executed. The House of Delegates took unprecedented action toward better hospital, medical school, and specialty relations which is for the primary benefit of patients and family physicians. This interim meeting went on record as favoring an all out campaign to halt hospital discrimination against general practitioners. Further, the A.M.A. will stimulate the formation of general practice departments in all medical schools and will insist that medical students be given experience in general practice. The Joint Commission on Accreditation of Hospitals will ask that full accreditation be withheld from community and general hospitals that exclude or restrict generalists on an arbitrary basis.

The panel discussion on "Psychotherapy in General Practice" was extremely interesting and informative. The highlight of this discussion was a letter from the moderator's (Dr. Forkner) pastor concerning his attitude on psychiatry. Here are a few quotes from the letter. "For a long time I have felt that people get off the beam in their thinking because they have followed the wrong guideposts leading them away from genuinely happy lives. If this is so, then we must erect for them different guideposts leading toward happy lives. . . . The Great Physician provided not only the formula for healing mental ills, but he also gave us precise tools to implement the formula. . . . The most powerful tool is prayer. . . . My plea is for a closer integration of psychiatry with spiritual values. . . . I would like to see a school of psychiatry founded on the theory that people think abnormally because they have never learned the formula for happiness and have not been taught how to apply this formula to their daily lives. . . . Herein may lie an enormous field of preventive medicine in which science and religion may share in a rich reward." Would you like to read this letter in its entirety?

In light of the above philosophical consideration, permit me to express myself concerning the wonderful experience our family and community recently enjoyed while we entertained our preceptee. Manohar Deosaransingh, Kansas medical school senior of Patna, Behar, India, really aroused us to the fact that America is now living with emphasis on artificial or material values. This should cause us to think seriously about our future as a nation, for in the beginning we were certainly founded on spiritual values. Where are we going, America?

Sincerely, in the Practice of the Art,



CONRAD M. BARNES, M.D., *President*

EDITORIAL COMMENT

Referrals to K.U.M.C.

The Committee on Medical Schools, of which Dr. R. W. Fernie of Hutchinson is chairman, believing that the mechanics of referring patients to the University of Kansas Medical Center are not completely understood, has obtained information from Dr. W. Clarke Wescoe.

Physicians are invited to use the faculty of the University of Kansas Medical Center for consultation services or for specialist care of patients should they care to do so. It should be quite clearly understood that this is a private arrangement between two physicians, just as any referral might be made elsewhere.

Patients cannot be accepted if referred to the medical center or if referred to a department. Referrals must be made to a specified physician. That, of course, can be done by mail or over the telephone, and a little history concerning the patient is as helpful to the professor at the school of medicine as it is under similar circumstances to the specialist in private practice. In return, the referring physician will receive a report concerning the patient, and, because of improved circumstances at the school, these reports will now arrive more promptly than in the past.

Listed below are the full time members of the faculty, divided into departments. It is recommended that this list be retained for future reference. Your committee wishes to remind again that a referral must be made to an individual physician and cannot be accepted otherwise. If there is any doubt concerning this procedure, the dean's office may be contacted and the communication will then be forwarded to the faculty member qualified to handle the problem.

The address of the University of Kansas Medical Center is 39th and Rainbow Boulevard, Kansas City, Kansas, and the telephone number is TAlbot 2-5252.

The Committee on Medical Schools hopes this information will be of service by increasing the efficiency of the normal professional contacts between the faculty and the Kansas physician in private practice.

DERMATOLOGY

Dr. Richard L. Sutton

MEDICINE

General

Dr. Mahlon Delp
Dr. Max Allen
Dr. Edward H. Hashinger
Dr. Robert Jordan
Dr. John Christianson
Dr. Robert Weber
Dr. Jesse Rising

Cardiovascular

Dr. E. Grey Dimond

Neurology

Dr. A. T. Steegmann

Metabolic

Dr. Robert E. Bolinger

Gastroenterology

Dr. Arthur Klotz

Hematology

Dr. Sloan Wilson

Chest

Dr. Martin FitzPatrick

OBSTETRICS AND GYNECOLOGY

Dr. L. A. Calkins
Dr. Robert Newman
Dr. Rosemary Schrepfer
Dr. Charles A. Hunter, Jr.
Dr. Eugene W. J. Pearce

OPHTHALMOLOGY

Dr. A. N. Lemoine, Jr.

OTORHINOLARYNGOLOGY

Dr. G. O. Proud

PATHOLOGY AND ONCOLOGY

Dr. Robert E. Stowell

PEDIATRICS

Dr. Herbert Miller
Dr. Herbert Wenner
Dr. Antoni Diehl
Dr. Franklin C. Behrle
Dr. C. Arden Miller
Dr. Daniel Darrow
Dr. Alice Marsh
Dr. Jacqueline Baumeister

PHYSICAL MEDICINE

Dr. Donald L. Rose
Dr. Edward B. Shires

PSYCHIATRY

Dr. William F. Roth, Jr.
Dr. M. T. Eaton, Jr.
Dr. Paul C. Laybourne, Jr. (Pediatrics)
Dr. Jane Tillinghast
Dr. Carol W. Powell

RADIOLOGY

Dr. Galen M. Tice
Dr. Donald R. Germann
Dr. Karl Youngstrom

SURGERY

General

Dr. Frank F. Allbritten, Jr.
Dr. Stanley Friesen
Dr. Creighton A. Hardin
Dr. C. F. Kittle
Dr. Paul Schloerb

Neurosurgery
Dr. William P. Williamson
Dr. Charles Brackett

Orthopedic Surgery
Dr. James B. Weaver

Plastic Surgery
Dr. David Robinson

Urology
Dr. William Valk

Medical Education Contributions

Announcement of the recent commitments of the Commonwealth Fund and the Ford Foundation in support of medical education has been generally received with acclamation and sincere public appreciation. Ten university medical schools have received grants totalling $7,150,000 for medical education from the Commonwealth Fund. The Ford Foundation will grant $90,000,000 to privately supported medical schools as endowment to help them strengthen their instruction, although the exact allocation to the individual schools has not yet been determined.

These two great philanthropic organizations have recognized that unless the increasing financial needs of medical education are met, the current high standards of medical training in the United States are in jeopardy. These grants will aid in meeting some of the needs that have existed during recent years, but they have not closed the gap that has been represented as being equivalent to approximately $10,000,000 annually. In other words, there is still great need for active and increasing support of medical education through the American Medical Education Foundation and the National Fund for Medical Education if the estimated current needs are to be adequately covered.

In explanation it's wise to stress that the Ford Foundation and the Commonwealth Fund have given these grants as *endowments.* At 4 per cent interest, this will return to the schools about $4,000,000 a year. This is a handsome sum, but it is still necessary to continue the good work that has been done to support our medical schools.

Longevity in Kansas

The Bureau of Medical Economic Research recently published a bulletin showing that Kansans are exceeding the national longevity rate. It was believed that this is of sufficient interest to be reprinted from the *Journal of the American Medical Association,* December 3, 1955.

"Often the opinion is heard that the healthiest people in the United States live in heavily populated areas where hospitals, physicians, and other health facilities and personnel are plentiful. Recently published data on the expectation of life, particularly at birth, in each of the 48 states for 1949-1951 refute this popular notion, especially for white males.[1] The statistical department of the Metropolitan Life Insurance Company has assisted the National Office of Vital Statistics of the Department of Health, Education, and Welfare in the preparation of life tables for each state. All the tables, including those for nonwhite males and females, will be published soon by the National Office of Vital Statistics.

"The six states with the highest expectation of life at birth for white males for 1949-1951 were: South Dakota, 68.4 years; Nebraska, Minnesota, and Iowa, 68.2 years; Kansas, 68.0 years; and North Dakota, 67.9 years. The expectation of life at birth for white males in the United States was 66.3 years. In 1939-1941 the six leading states for white males were Nebraska, South Dakota, Minnesota, Iowa, North Dakota, and Kansas. Thus it is obvious that white males of these sparsely settled states still enjoy comparatively low mortality. The only changes apparent in the figures cited above are (1) South Dakota has replaced Nebraska as the leader, and (2) Kansas and North Dakota have exchanged ranks.

"It is not possible to say that longevity will actually be greatest among the white males of these six states, because expectation of life is an actuarial computation based upon the mortality rates at all ages in three specified calendar years. The computation, strictly speaking, is not a forecast of the mortality that will prevail during the entire lifetimes of the babies born in each of these six states in the specified three calendar years. These favorable mortality records among white males in these six states must be attributed, in part, to the strong North European strains in the population and favorable selection factors in migration. The number of miles that many people in these states must travel to visit a physician or become a patient in a hospital might be considered a health hazard by many urban dwellers, but the people in the West North Central region of the United States have developed a way of life that makes them well adjusted to these spatial conditions.

"Nebraska retained its leadership in life expectation at birth for white females with 74.0 years as compared with 72.0 years for the entire United States. Oklahoma, Florida, Iowa, Kansas, and South Dakota follow with 73.8 to 73.6 years. A more general observation is that the state variations in longevity as indicated by expectation of life at birth were less in 1949-1951 than they were in 1939-1941. The leaders in expectation of life at age 25 were South Dakota,

[1] State Variations in Longevity, Metropolitan Life Insurance Company Statistical Bulletin, Oct., 1955.

Nebraska, and Iowa, with 46.8 years for white males. The leading states for white females at age 25 were Oklahoma, Nebraska, Florida, Arkansas, Kansas, Iowa, Texas, South Dakota, and Utah, ranging from 51.8 years to 51.1 years. The states in which the people at age 65 had the greatest expectation of life were Arkansas, Florida, and Oklahoma for white males and Florida, Arizona, Arkansas, Oklahoma, and Texas for white females.

"The variations in expectation of life in 1949-1951 at these three selected ages were less marked, of course, among the nine regions than among the 48 states. The West North Central region (Minnesota, North Dakota, South Dakota, Iowa, Nebraska, Missouri, and Kansas) had the highest expectation of life for white males at birth (67.8 years) and at age 25 (46.4 years), but the West South Central region ranked first at age 65 (13.4 years versus 13.3 years). For white females the West North Central region had the highest expectation of life at birth, 73.3 years; at age 25 its 50.9 years ranked it second to the West South Central region with 51.2 years; at age 65, however, it was outranked by a small margin by the West South Central, Mountain, and Pacific regions. In 1939-1941, the West North Central region ranked first in expectation of life at birth, at age 25, and at age 65 for white males; and first at birth, first at age 25, and tied for second at age 65 for white females. These data for the nine regions, as well as those for the individual states, should remove all doubt about where the expectation of life is and has been the highest, particularly at birth and at age 25."

Workmen's Compensation

A new book has recently been published by the Workmen's Compensation Commission of Kansas. It includes the Workmen's Compensation Law as well as procedure, rules, and theory of compensation.

The Committee on Industrial Medicine of the Kansas Medical Society, of which Dr. W. L. Anderson of Atchison is chairman, played a part in the publication by studying the medical fee schedule and recommending many changes from the old schedule. The commission accepted the recommendations, and the fees published are those outlined by the committee.

In discussing the entire structure of workmen's compensation, and particularly the physician's report, the following suggestions are made to expedite administration of the program and to continue the good relationship between the medical society and the Workmen's Compensation Commission. Most physicians are familiar with the physician's report blank. Sample copies may be obtained by writing to Workmen's Compensation Commission, 501 Jackson Street, Topeka. The suggestions follow:

1. When the word "member" is used, it refers to the general body disability as well as fractures, amputations, etc.

2. A separate x-ray diagnosis attached to the report blank is accepted and recommended.

3. Space is provided to state whether the patient needs further medical care and for how long. If the patient needs additional care beyond stated date, the physician should report this in writing to the employer or insurance carrier.

4. With reference to eye injury, the physician may write a letter and attach it to the form, stating percentage of loss of vision due to the accident.

5. The physician should not mention illnesses which do not pertain directly to the injury unless the disability has been aggravated by the injury.

6. According to the law, the physician must submit the report to the employer or insurance carrier within 15 days after examination.

Questions in regard to workmen's compensation may be directed to the Workmen's Compensation Commission or to the Committee on Industrial Medicine of the Kansas Medical Society.

Statistics on Congressional Bills

A study of bills introduced in the last two sessions of Congress, the 83rd and the 84th, made by the A.M.A. Committee on Legislation, shows that an increasing number of subjects considered have medical implication.

During the two sessions of the 83rd Congress, 16,470 bills were introduced, and 407 of those pertained to medicine. During the first half of the 84th Congress, 11,914 measures, 403 pertaining to medicine, were introduced.

The A.M.A. committee has met four times so far this year and has considered 284 bills embodying 92 major legislative proposals. The group has also considered 10 major policy questions on which legislation has not yet been introduced. Fifteen witnesses have testified before committees of the Congress on 21 separate bills, and the secretary and general manager of the A.M.A., Dr. George F. Lull, has submitted the written views of the association on nine other proposals.

All of the 403 measures introduced this year were reported in the A.M.A. *Washington Letter*. Congress held hearings on 49 of those, and the committees reported favorably on 11. Five were passed by both houses and have been signed by the President. Four of the five that became law were supported by the A.M.A.

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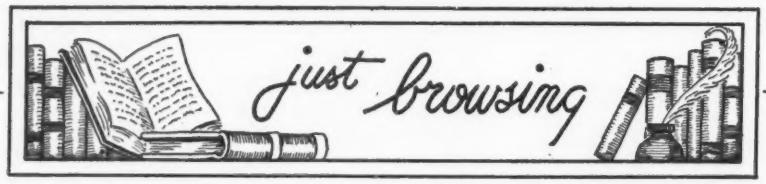

". . . The causes [of morning sickness] are increased activity of the nerve force, whereby the equilibrium between digestion and assimilation is greatly disturbed. With those women who have habitually weak digestion this is not much of a symptom. Indeed, they will have better digestion and keener appetites for the reason that the action of the nerve force has been increased, whereas it had previously been deficient. When this annoying sickness is persistent and obstinate, yielding to no remedies, diet or regimen, we may suspect displacement of the womb, possibly ulceration at its neck. . . .

"Among women who live in towns and lead a sedentary life constipation is a frequent attendant of pregnancy. There are other things besides indolent habits that may give rise to constipation, among which are the pressure of the enlarged womb upon the bowels, and a kind of torpor produced by the diverting of the nerve force and muscular supply to the womb.

"When there is a general swelling which is more immediately noticeable in the hands and face, it may . . . be caused by a disorder known as albuminuria. This is a condition in which the albumen of the blood, instead of going to supply the body, finds its way out through the kidneys. Such swellings should lead at once to an examination of the urine to determine whether or not albumen is present. . . . Indeed, it is a wise precaution to use this test at different times during pregnancy, whether the disorder under discussion is suspected or not. The violent and fatal convulsions in childbed are occasioned by albuminuria, which is induced by a condition of the kidneys that obstructs the circulation of the blood, causing a retention of urea in the blood, and thus producing what is known as uraemic poisoning. This condition exists, to a greater or less extent, oftener than is suspected by most people, including physicians; hence the urgency of frequent tests to decide about its existence. It can be corrected by a skillful physician. . . .

"Abortion must be regarded as a serious evil. It not only deprives the mother of the product of her pregnancy, but often places her health and even her life in peril. . . . Fleshy women subject to profuse menstruation, very impressible, nervous women easily excited by passion or mental disturbances, and those who indulge immoderately in the pleasures of society, dancing, late hours and tight lacing, are liable to the misfortune. Such as are occupied at the sewing machine are constantly exposed to this mishap. . . . A sudden leap from a carriage, reaching up and straining, excessive fatigue, too frequent coition, violent fits of anger and other passions, are causes.

"Under such conditions [flooding] the woman's head should be lowered and the hips be a little elevated. An injection of ice-cold water will often be effectual; at the same time some pieces of ice may be swallowed. A treatment quite the reverse of this is popular and efficacious at the present time, and consists in using hot-water injections, temperature 95° to 100°, and applying rubber bags filled with hot water to the spine. Place the child to the breast. The object of these various means is to promote contraction of the womb. Nothing is simpler or perhaps better than dipping the hand in cold water, placing it on the abdomen, and grasping the womb to stimulate it to contract. . . ."

Thus did they explain and treat some of the complications of pregnancy 70 years ago, as detailed in a *Compendium of Health* of the period. Explanations of symptoms and the treatments prescribed sound peculiar to us today, yet there are within the same paragraphs statements which are as acceptable today as they were then—for example, that "morning sickness may begin almost immediately after conception" but "generally does not begin until after the lapse of two or three weeks, but then continues more or less constantly and severely for three or four weeks . . ." and that "miscarriages happen oftener at the third month of pregnancy." How do you suppose our present books will sound in 2025?—*O.R.C.*

Tumor Conference

Basal-Cell Carcinoma of the Lower Lip

Edited by HOWARD P. FINK, M.D.

Dr. Friesen: The following case is presented because it concerns a common lesion occurring in an uncommon location. Will you give us the history, Dr. Gwinn?

Dr. Gwinn: This patient is a 75-year-old retired farmer who, one year ago, developed a sore on the right side of his lower lip which failed to heal. He tried various home remedies, none of which seemed to do any good. The sore didn't cause him any trouble and did not enlarge. However, in August, two months ago, he had a very painful swelling in the right submandibular area, just below the lesion on his lip, for which his local doctor referred him here.

The workup here revealed the sore on his lower lip, which you see as a punched out, indurated ulcer about .5 cm. in diameter (Figure 1). The right submandibular area is quite swollen and painful. A biopsy of the lesion of the lip was reported as a basal cell carcinoma. X-ray examination showed rarefaction of the mandible and abscesses of the teeth in the swollen area but no evidence of any metastatic disease. The teeth were subsequently removed, and the submandibular abscess and cellulitis were improved after penicillin and drainage.

Figure 1: Ulcerated lesions of the lip and submandibular area.

It is interesting to note that the patient's father died of a carcinoma of the lip.

Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute, U. S. Public Health Service, and from the Kansas Division of the American Cancer Society. Dr. Fink is a Trainee of the National Cancer Institute.

Dr. Friesen: This looks like a serous lesion. May we see a microscopic section of the biopsy?

Dr. Mantz: Two pieces of tissue were taken. Both of them show a typical basal cell carcinoma consisting of large club-like clusters of cells (Figure 2) which penetrate the underlying connective tissue to a considerable depth. The epidermis is slightly acanthotic except over the lesion where it becomes thin and ulcerated. Numerous small cyst-like areas are scattered throughout this neoplasm, but otherwise it appears to be a fairly orderly basal cell lesion with distinct palisading of cells at the periphery. The clear areas sometimes contain foamy-appearing cells, probably macrophages. They suggest, however, the remote possibility that this lesion may be of cutaneous adnexal origin, possibly derived from the sebaceous glands.

This lesion shows, as most basal cell carcinomas do, the defense mechanism of the host against the advancing edges of the carcinoma, in the form of an inflammatory reaction, which, in this lesion, includes giant cells of the foreign body type.

Material from the mandibular area shows chronic inflammation consistent with the lining of an abscess.

Dr. Friesen: May we see the x-rays?

Dr. Goertz: X-rays of the mandible show abscesses at the roots of the teeth and decalcification

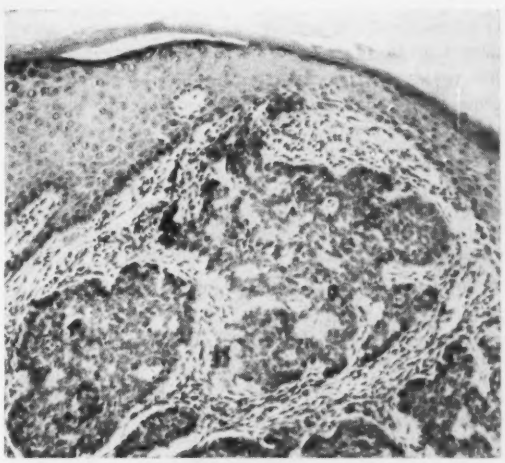

Figure 2: Basal cell carcinoma, showing downgrowths of neoplastic epithelium containing cystic spaces. x 100.

which seems to be a little more marked on the right than on the left.

Dr. Friesen: Dr. Robinson, when I went to medical school, we were taught that basal cell carcinomas occur above the mouth-to-ear line, and squamous cell carcinomas below. This case is apparently an exception to that rule.

Dr. Robinson: That's right. If one looks at this lesion and at the mass in the patient's neck, which seems to be fixed to the mandible, one might say that this is a bad situation. Here, apparently, is a carcinoma which has metastasized and is now suppurating and necrotic, and surgery would have to be radical to save this man's life. There should be something in the history, though, that would lead us to think that our first impression is mistaken. Dr. Gwinn, how rapidly did this mass develop in his neck?

Dr. Gwinn: Very rapidly. It appeared in the course of a few days.

Dr. Robinson: Was it tender?

Dr. Gwinn: Yes.

Dr. Robinson: Did it drain spontaneously?

Dr. Gwinn: Yes. It was draining when he came in.

Dr. Robinson: This story indicates an abscess rather than a metastasis. Still, one can't be certain without more data. The biopsy here was of primary importance. To find a basal cell carcinoma by biopsy in an ulcer of the lower lip is surprising because, as Dr. Friesen indicated, nearly all such lesions are squamous cell carcinomas, which tend to metastasize, whereas basal cell lesions rarely do. More basal cell than squamous cell lesions occur in the upper lip, and carcinomas of the lower lip are nearly all squamous. Occasionally, we do see each of these lesions in the opposite location.

When Dr. Edwards removed several carious teeth from this man's lower jaw, he found extensive periodontal infection. So, I think we can say that the jaw lesion is not a metastasis but simply a local infection from bad teeth, and our initial impression that this was a metastasizing squamous cell carcinoma was wrong.

The question of treatment resolves itself now to simple excision of the lesion. We will do this in a few days, when the inflammatory edema has subsided somewhat.

Dr. Friesen: Is he getting antibiotics?

Dr. Gwinn: Yes, he is.

Dr. Friesen: Is the swelling subsiding?

Dr. Gwinn: Yes.

Dr. Friesen: Are basal cell carcinomas usually treated by surgery or by irradiation, Dr. Robinson?

Dr. Robinson: We prefer to treat them by surgery because we think that we have better control of their removal than do the radiologists. At the time of removal, if we are at all in doubt, we can have frozen sections made to be sure that we are dealing with a tumor. Also, we believe that by not obscuring the issue with late irradiation changes and by eliminating radiation fibrosis, we can diagnose recurrences a little quicker. It is true that we usually must cut 5 to 10 mm. beyond the visible edge of the lesion to be sure of its complete removal. Paraffin sections must be carefully studied to be sure that we have cut beyond any crab-like extensions of the tumor, but, in general, where one can do so without distorting tissues to a great extent, and where the lost surface can be easily replaced, I think it is better to excise such lesions and be done with them.

However, there are some parts of the face where we prefer irradiation because we think the radiologists do a better job than we do with less distortion and mutilation. This is a moot question, and some people would elect to excise all basal cell lesions, but I am sure the little ones around the eyelids and inner canthus where they can be well shielded, and those around parts of the nose not directly over cartilage, can be well handled by irradiation.

Dr. Friesen: If you had a patient with a large ulcerated lesion on the cheek which would necessitate a skin graft if you excised it, would you prefer excision to irradiation?

Dr. Robinson: Yes, I would, because I think that I would have better control of the tumor. However, when a large surface defect is left after excision, one must figure how he is going to cover the wound, since wounds of the face are rarely left open for long. A skin graft may be required. The choice between irradiation and surgery often depends on the ability and preference of the man who is treating a particular lesion. I am sure that some of the lesions that I would excise, Dr. Tice and his associates could treat with equally good results.

Dr. Tice: I am sure that both radiologists and surgeons are influenced by their personal experience in treating epitheliomas of the skin. As a radiologist, I am not particularly concerned whether a given lesion is a basal cell or squamous cell carcinoma, but I am concerned about the location of the lesion and, in some instances, about the size of it. The common site of a basal cell lesion is the face, and here small cancers can be treated easily, effectively, and with good cosmetic results by irradiation. If the lesion involves an entire cheek or the entire forehead, the radiologist should consider calling for surgical consultation, although even such large lesions may be treated in sections using deep x-ray therapy instead of superficial therapy. A lesion over cartilage or on the back of the hand, however, is almost always referred to the surgeon because of the danger of dam-

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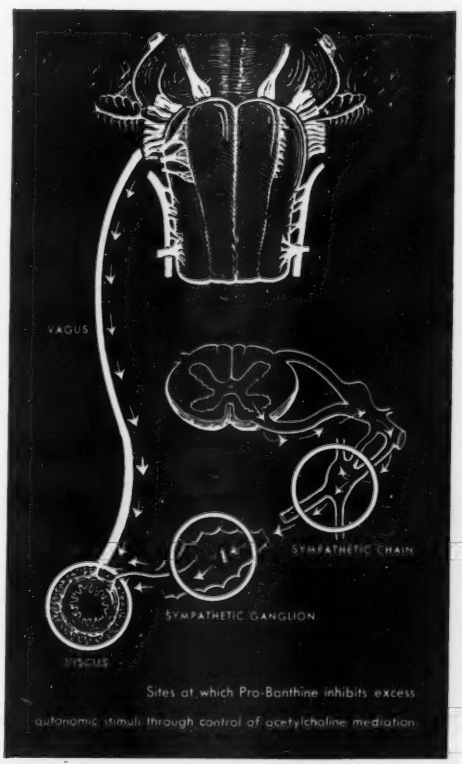

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.
2. Roback, R. A., and Beal, J. M.: Gastroenterology *25*:24 (Sept.) 1953.

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aging adjacent tissues with the dose of x-ray required for treatment.

I would agree that many small basal cell cancers can be treated just as well by the surgeon as by the radiologist and perhaps treated more easily. I think the scar resulting from surgery is usually more obvious than that produced by the radiologist. With either treatment, a cure may be expected for most lesions.

Student: Dr. Robinson, would you biopsy a carcinoma of this sort before you treat it, or would you excise it and call the excision the biopsy?

Dr. Robinson: Whenever the lesion is small and we are reasonably sure what it is, we prefer to do an excision biopsy and be done with the matter at once. But when there is doubt about the diagnosis, as there is in this case, it is better to establish the diagnosis by biopsy, even at a time when there is fear of spreading the tumor, because the difference in treatment can be marked. However, there are instances in which we elect not to do incision biopsies.

For example, by aspiration biopsy of tumors in the parotid region, we have been able to prove the diagnosis often enough to think that we are justified in using this procedure. Again, if one suspects a melanoma, one doesn't like to do an incision biopsy for fear of spreading the cells via the blood stream. When the differential diagnosis rests between basal cell and squamous cell carcinoma, I think it is justifiable to take a piece off the side, including some normal tissue, so that sections will show what type of cells are growing actively at the edge of the lesion. In this patient I think biopsy was certainly justified; if we had thought that this was a squamous cell carcinoma, we would have assumed that the lesion on the jaw probably was a metastasis and our treatment would have been in error.

Dr. Johnson: I think that if the information that is obtained from biopsy will alter the choice of treatment, then biopsy should be done. At the Ellis Fischel Hospital, we don't hesitate to biopsy a suspected melanoma because, if the lesion proves to be a melanoma, the treatment will be different from that for a basal cell or squamous cell carcinoma. If the lesion is small enough, we do an excision and primary closure; but if it is large and would require a deep incision for removal, a piece is taken from the edge.

Dr. Friesen: This, at least, is one case in which initial impressions are misleading and treatment might have been more radical than necessary if the diagnosis had not first been established by biopsy. This case illustrates the fallacy of assuming that two lesions, occurring in the same patient at the same time, are necessarily caused by a single disease.

Chamber of Commerce Policy

A study of medical care for veterans was advocated by the United States Chamber of Commerce at the close of its recent meeting. Policy declarations of the organization are published in book form, and the book this year, in a chapter headed "Economic Security," includes the paragraph below.

"The federal government provides for the medical care of veterans through federally owned and operated hospitals and salaried physicians. The rapid expansion of the veteran population and the extent to which services are rendered to veterans with non-service connected disabilities justify a searching re-evaluation of the entire program. A definition of 'beneficiary' that is just both to the veteran and to the nation as a whole is urgently needed."

Television Programs on the Eye

Two new television "script-with-film" programs featuring current health education information on the eye and its functions have been released by the A.M.A. for use by county medical societies. Requests for the films and scripts are to be addressed to the A.M.A. Television Film Library.

"A Clear Picture" is the title of the program devoted to the structure of the eye, and "Wonderful Spectacle" describes the functions of glasses and lenses. Either can be used as a separate 15-minute program or the two may be combined for a single presentation. Dr. Brittain F. Payne, New York ophthalmologist, is the film demonstrator.

Grants for Heart Research

In the first decade of its existence the Life Insurance Medical Research Fund has given more than $7,000,000 for heart research, according to a report issued recently by the Institute of Life Insurance. Awards for 1955 total $929,400 and include $5,500 to the University of Kansas School of Medicine for research by Dr. Kenneth E. Jochim on effects of arteriosclerosis on arterial circulation.

What is to happen to the derelicts? Although tuberculosis occurs in any type, level or stratum, there are people especially prone—the dispossessed and the defeated, the disintegrated and the defenseless. The teacher who cannot get a job because of some x-ray shadow; the young man excluded from industry; the man who finds he cannot enter any superannuation scheme because of a past history of tuberculosis—what are we to do with them?—*Harley Williams, M.D., Nat. Tuberc. A. Tr., May, 1954.*

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PHYSICIANS' ACTIVITIES

Dr. Paul H. Lorhan, of the University of Kansas Medical Center, was an instructor on the subject of "Geriatric Anesthesia" at a November meeting of the American Society of Anesthesiology in Boston. He also participated in two panel discussions.

Dr. Glen Q. Street, Jr., Wichita, has been appointed part-time medical and psychiatric consultant for the Wichita Recovery Center of the Kansas State Commission on Alcoholism.

The Kansas Division of the American Cancer Society announces that **Dr. J. P. Berger,** Wichita, is now serving as its president.

Dr. Mary T. Glassen, Phillipsburg, was recently re-elected president of the Kansas Council of Children and Youth.

The appointment of three new health officers was recently announced: **Dr. Marita Scimeca,** Kiowa, for Barber County; **Dr. James E. Henshall,** Osborne, for Osborne County, and **Dr. Willard F. Werner,** Tribune, for Greeley County.

Dr. George W. Jackson, director of state institutions, Topeka, was recently named to membership in the Central Neurology and Psychiatric Association.

Dr. Richard F. Looker, Wichita, addressed the Hillsboro Kiwanis Club last month on the subject of "Advances in the Diagnosis of Heart Disease."

Dr. Frederick A. Gans, Salina, has been elected to membership in the American Academy of Pediatrics.

Dr. W. G. Cauble, Wichita, is the author of a paper, "Acute Abdominal Pain in Polycythemia," published in the October issue of *American Surgeon.*

Dr. William Aldis, who has been practicing in Corpus Christi, Texas, since his release from the Navy, has joined the staff of the Newman-Young Clinic in Fort Scott. He is a brother of **Dr. John Aldis** and **Dr. Henry Aldis,** also of Fort Scott.

Dr. Austin J. Adams spoke on "Functioning in the Field of Mental Health" at a recent meeting of the Wichita Lions Club.

Dr. William L. Valk, of the University of Kansas Medical Center, participated in a panel on renal lithiasis at a meeting of the American College of Surgeons in Chicago in November.

A talk on the diagnosis of different kinds of tumors was made to the Emporia Kiwanis Club recently by **Dr. Walter E. Luedtke,** Emporia.

Dr. Tom Taylor, formerly on the staff of the sanatorium at Norton, began a residency in internal medicine at the Veterans Hospital in Denver on January 1.

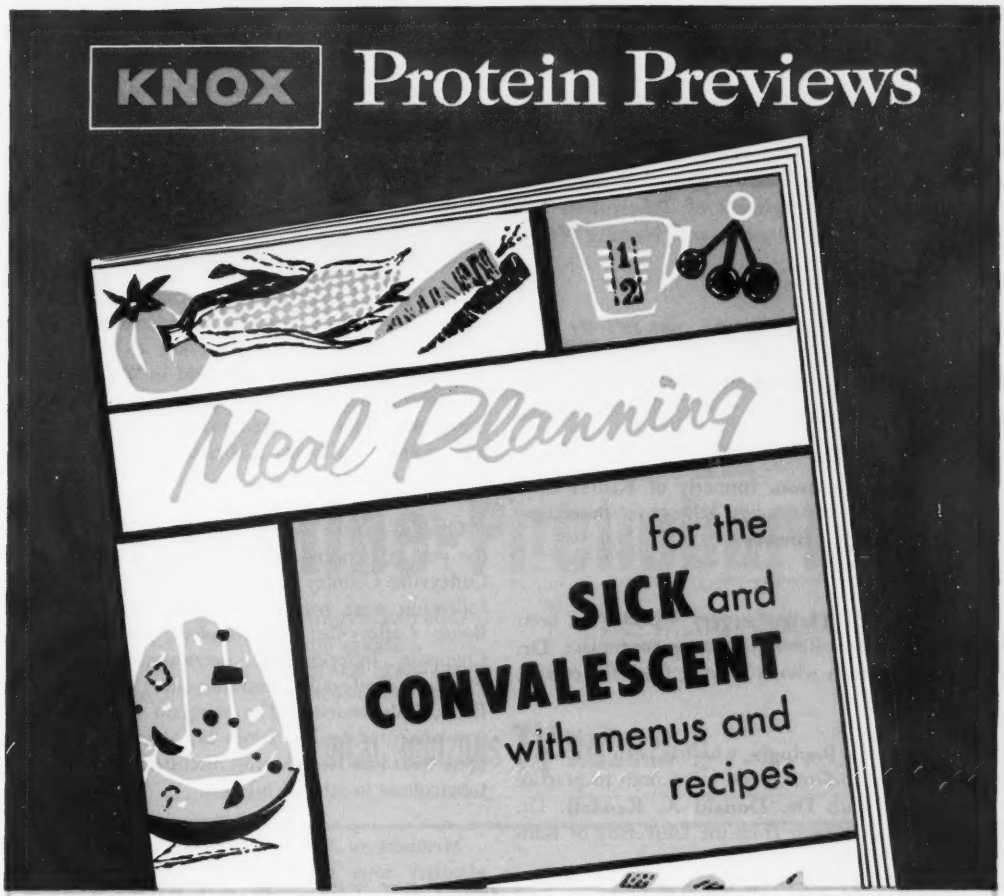
Dr. V. J. Brown was discharged from the Air Force last month and has returned to his practice in Wichita.

Dr. Richard H. Claiborne, who has been on the staff of the Parsons State Training School during the past six months, has moved to Baxter Springs and is establishing a practice there. He was graduated from the University of Kansas School of Medicine in 1953, served his internship at William Beaumont Army Hospital in El Paso, and then spent a year as a medical officer at the White Sands Proving Ground, New Mexico.

Two Wichita physicians, **Dr. Mary J. Blood** and **Dr. William F. McGuire,** recently became fellows of the American Academy of Pediatrics.

Dr. Robert E. Bodmer, who formerly practiced in Coldwater, is now taking a year's residency in internal medicine at the University of Kansas Medical Center.

Dr. John L. Lattimore, Topeka, spoke on methods of diagnosing cancer and leukemia at a meeting



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of doctors of the Hiawatha community on November 30.

Dr. John Fulton and **Dr. Alfred Tocker,** Wichita, were speakers at a meeting of registered nurses held in Wichita last month under the sponsorship of the Wichita-Sedgwick County Health Department. The subject was "Management of Tuberculosis Patients from Medical, Surgical, and Nursing Viewpoints."

Dr. Karl Menninger, Topeka, was recently honored by the National AmVets organization when he was presented its fourth annual rehabilitation award for his service to veterans.

Dr. William Dodson, formerly of Kansas City, Missouri, is now practicing in Ulysses in association with **Dr. Marshall Brewer.**

Dr. Charles E. Henneberger, Atwood, has been named coroner of Rawlins County to replace **Dr. Kenneth L. Knuth** who is moving from the county.

Dr. Charles B. Replogle, who has been practicing in Ellis, moved to Great Bend last month to practice in association with **Dr. Donald A. Kendall.** Dr. Replogle was graduated from the University of Kansas School of Medicine in 1952.

Dr. Edgar Johnson, who formerly practiced in Kansas City, Missouri, has opened an office in La-Cygne.

Dr. Harlan W. Berthelsen, who formerly practiced in Moline and McPherson, is now practicing in Howard.

COUNTY SOCIETIES

Dr. Charles S. Joss will serve as president-elect of the Shawnee County Medical Society as the result of an election held in Topeka on December 6. Dr. Clyde B. Trees, who took office as president at that meeting, will be assisted during the coming year by the following: Dr. Dwight Lawson, vice-president; Dr. Clovis Bowen, secretary; Dr. James McClure, treasurer. Dr. Dale Dickson has begun a three-year term on the board of directors, as has Dr. Lucien R. Pyle on the board of censors. Two, Dr. William O. Martin and Dr. Robert H. O'Neil, have started two-year terms on the medical service board.

The meeting, at which the doctors' wives were guests, included a dinner, a floor show, and dance. Membership in the 85-50 club, composed of physicians who have reached 85 years of age or have been in practice for 50 years, was conferred on Dr. C. S. Smith of Rossville and Dr. W. W. Reed.

Dr. Frank F. Allbritten, Jr., chairman of the department of surgery at the University of Kansas Medical Center, was guest speaker at a meeting of the Sedgwick County Society held in Wichita on December 6. His subject was "Cancer of the Lung." In the afternoon he conducted a clinic at St. Joseph Hospital with Dr. Edward S. Brinton and Dr. John G. Shellito as moderators.

Officers of the Montgomery County Society for the year 1956 were elected at a meeting held at the Coffeyville Country Club on November 16, and the following were named: president, Dr. William R. Beine, Coffeyville; vice-president, Dr. William G. Chappuie, Independence; secretary, Dr. Virgil E. Flanders, Coffeyville, and treasurer, Dr. Gerald C. Bates, Independence. The program for the evening was presented by Dr. James M. Mott, of the Kansas State Board of Health, who discussed skin testing for tuberculosis in school children.

Members of the Labette County Society and its auxiliary were guests of Dr. and Mrs. I. Joseph Waxse, Oswego, at a dinner at the Waxse home on November 9. Separate meetings were held later, and the speaker for the doctors' session was Dr. R. L. Ferguson, Joplin, who discussed "The Pathology of Lymph Nodes." The group elected the following officers for 1956: president, Dr. Arthur P. Burgess, Oswego; secretary-treasurer, Dr. V. L. Jackson, Altamont.

Thirty members of the Central Kansas Society were present at a meeting held in Russell on November 17. During the afternoon session Dr. John F. Thurlow, Hays, and Dr. Richard L. Dreher, Salina, presented scientific papers. A business session was held after a dinner, and the following officers were elected: president, Dr. John C. Artman, Hays; vice-president, Dr. Frank A. Dlabal, Wilson, and secretary-treasurer, Dr. Rex C. Belisle, Hays.

Dr. Donald E. Ray, Chanute, was elected president of the Neosho County Society at a meeting held at the Tioga Hotel, Chanute, on November 30. Dr.

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Edward E. Long, Humboldt, was named secretary-treasurer. The program for the evening, following a dinner meeting with the Auxiliary, consisted of a paper on mediastinal tumors by Dr. Vernon Berkey, Pittsburg.

Approximately 100 persons were present at a Christmas party for members of the Southeast Kansas Medical Society and their wives at the Columbus Country Club on December 8. Dr. Clarence H. Benage, Pittsburg, gave a short talk, after which a floor show was presented. The dinner menu was made up in medical style with scientific terminology for the different items served.

The regular meeting of the Wyandotte County Society was held at the Red Cross Building in Kansas City on December 20. Dr. Alvin Silvers spoke on "Hospital Pharmacies."

Dr. Robert O. Brown was elected president of the Atchison County Society at a meeting held at Atchison on December 6. The following were chosen to assist during 1956: vice-president, Dr. Arthur Whitaker; secretary-treasurer, Dr. Wayne O. Wallace; delegate to state meeting, Dr. Edwin T. Wulff; alternate, Dr. Wallace.

THE KANSAS PRESS LOOKS AT MEDICINE

Editor's Note. In this section the JOURNAL reproduces editorials relating to medicine which have appeared in the lay press. An effort is made to include both favorable and unfavorable comments, and the Editorial Board in no instance assumes responsibility for the opinions expressed.

BACKWARD, TURN BACKWARD

To one whose memory goes back to the turn of the century or a little before, the harangue of the pitch men on radio and television as they hawk their medicinal concoctions is very similar to the old-time medicine shows which peddled nostrums guaranteed to cure most everything with which human beings are afflicted.

That was away back when Old Dr. Munyon, with his index finger pointing upward, Kickapoo Indian Sagwa, Payne's Celery Compound, and Sloane's Liniment, good for man or beast, was apt to be found in every medicine chest. In every drug store were Pink Pills for Pale People—now called vitamins and "guaranteed to make you feel better fast"—Glover's Hair Restorer, Sweet Caporal cigarettes with their risque pictures, snuff, flaxseed poultices and Lydia Pinkham's Compound. The latter was for womenfolks and its use was not fully understood by the boys who wondered why it was the only medicine with which they were not rubbed or dosed.

The Winchester calendars were in thousands of homes. Two big companies had a monopoly with Star and Horseshoe spitting tobacco until old Battleax plug came along and almost cornered the market with its cut price. For domestic treatments mom gave the kids pumpkin seed tea for worms, tied an old sock around their necks for throat, put an onion bag on their chests for croup and tied a bag of asafoetida about their necks to keep infectious diseases away.

Other standbys were Ayers Cherry Pectoral, good for whooping cough, croup, sore throat, colds and lung trouble peculiar to children. Dr. Owens Electric Belt, with its mixture of copper and zinc plates, was a cure for rheumatism, lumbago, nervous debility, sexual exhaustion and indiscretions. Piso's Cure for Consumption—now called tuberculosis—was a sure cure and could be administered without the help of a TB association, while Ely's Cream Balm was a cure for catarrh, hay fever, cold in the head and deafness, and cost only 50 cents. On the label of DeWitt's Little Early Risers was the assertion that "If you are not bright, these pill will make you so."

That was before the days of the medical specialist and a doctor did everything for $2, win, lose or draw. But $2 could be an extravagance then and no doctor was called so long as the patient was conscious and had been rubbed with so many things that he was liniment-logged when the doctor arrived.

One hesitates to think what the old-time doctor (?), with his Prince Albert coat, his stove-pipe hat and his long black string tie, standing on the platform of his free medicine show with a smoking kerosene torch at each corner, could do today if he could make his pitch over the radio or television. But be it said to his credit, he didn't act like a confidence man, put on a synthetic smile and wheedle his way into your home with a lota bull, bunc and baloney. —*F. J. C., Kingman Leader-Courier, December 2, 1955.*

NEVADA SOCIETY INSURES ATTENDANCE

A measure to increase attendance at its meetings was recently adopted by the medical society in Washoe County, Nevada, in the form of a by-law applying to all physicians on its roster. It reads, "Each doctor shall attend 50 per cent of the meetings of the Washoe County Medical Society, with the exception of doctors who are 50 years of age or older with 10 years attendance at the meetings, or 55 years of age with five years attendance." Penalty for nonattendance is $100 with the possibility of suspension or expulsion for two consecutive years of nonattendance.

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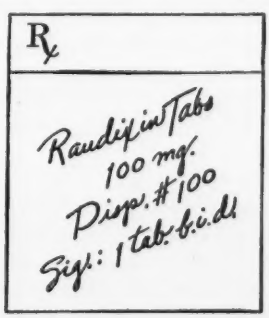

*Ataractic, from ataraxia: calmness untroubled by mental or emotional excitation. (Use of term suggested by Dr. Howard Fabing at a recent meeting of the American Psychiatric Association.)

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DEATH NOTICES

THOMAS GROVER ORR, SR., M.D.

Dr. Thomas G. Orr, 71, professor emeritus at the University of Kansas School of Medicine, died on November 19 after suffering a heart attack. From 1924 to 1949 he had served as professor of surgery and chairman of the department of surgery at the school.

A graduate of Johns Hopkins University School of Medicine in 1910, Dr. Orr came to this area in 1915 and engaged in private surgical practice before affiliating with the medical school. He was a diplomate of the American Board of Surgery, a fellow of the American College of Surgeons, and a member of the American Surgical Association and the Western Surgical Association. He also served as editor of *American Surgeon*. During World War II he was an officer in the Army medical corps.

RAYMOND JOSEPH LEIKER, M.D.

A coronary occlusion on November 22 brought death to Dr. Raymond J. Leiker, 52, at his home in Great Bend. A member of the Barton County Medical Society, Dr. Leiker had practiced in Great Bend since 1937. He was graduated from St. Louis University School of Medicine in 1929 and began practice in Ellinwood in 1931, remaining there until 1937. He served in the Army medical corps during World War II.

BERT ELBA MILLER, M.D.

Dr. B. E. Miller, 75, a member of the Morris County Society, died at Council Grove on November 26. Had he lived until spring, he would have completed 50 years of practice in Council Grove, and the community was planning a "Dr. Miller Day" in his honor. He was a graduate of the University Medical College of Kansas City with the class of 1905.

RALPH R. CLUTZ, M.D.

Dr. Ralph R. Clutz, 78, who had practiced for 55 years, died at an Atchison hospital on November 30 after an extended illness. He was graduated from Kansas City Medical College in 1900 and began practice in Bendena in 1902, remaining there until his illness. He had served two terms as health officer for Doniphan County. Dr. Clutz was an honorary member of the Doniphan County Medical Society.

LOUIS BOUCHER GLOYNE, M.D.

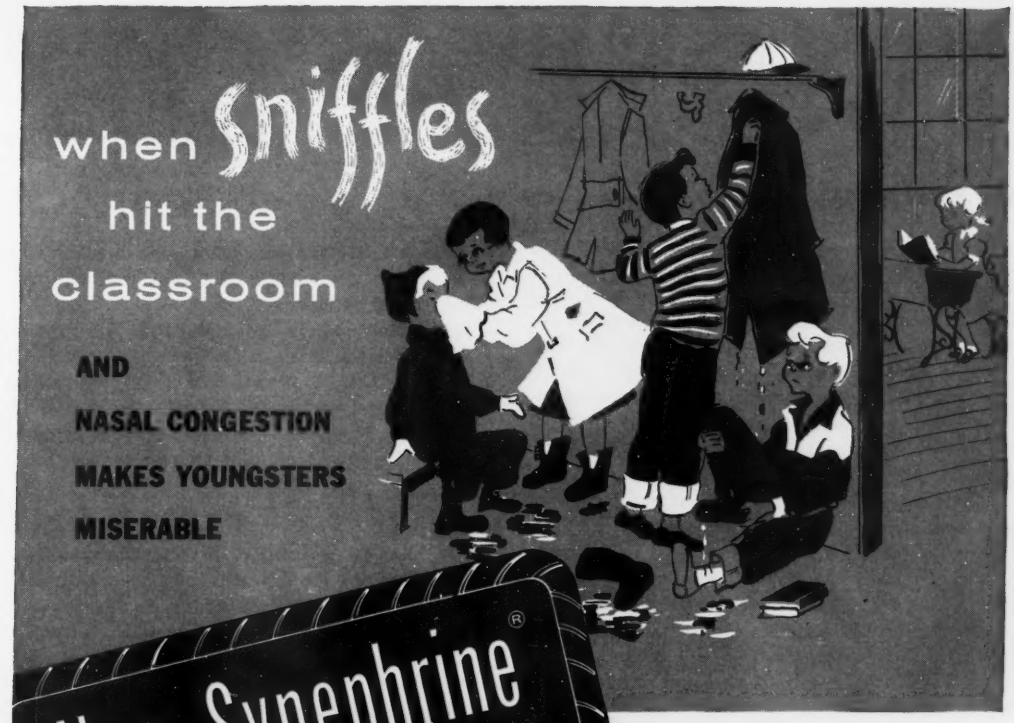
Dr. Louis B. Gloyne, 62, a physician in Kansas City for 37 years, died at Bethany Hospital there on December 8. He was graduated from the University of Kansas School of Medicine in 1918, served his internship at St. Margaret's Hospital in Kansas City, and then began private practice. In 1926 he went to Vienna for a postgraduate course in surgery.

Dr. Gloyne was active in medical society affairs throughout his professional life. He had served a term as president of the Wyandotte County Medical Society, of which he later became an honorary member, had been on the Council of the Kansas Medical Society, and had been a member of various committees. He was one of the organizers and a past president of the Kansas Academy of General Practice.

A son, Dr. Howard F. Gloyne, Kansas City, is among the survivors.

CHARLES HENRY LERRIGO, M.D.

Dr. C. H. Lerrigo, 83, an organizer of the Kansas Tuberculosis and Health Association, died in Sylacauge, Alabama, on December 4. He was born in England and came to Kansas in 1886. In 1900 he was graduated from the Homeopath Medical College in St. Louis, and in 1901 he opened an office in Topeka. His career in public health work began in 1905 when he was appointed to the Kansas State Board of Health, and he continued in that capacity for 18 years. The Kansas Society for the Study and Prevention of Tuberculosis was organized under his direction, and he served as one of its officers from 1908 to 1919. During World War I he served overseas as commanding officer of the Washburn Ambulance Company with the rank of major. In 1922 he became secretary of the Kansas Tuberculosis and Health Association, continuing in that position until his retirement in 1947 and as editor of *Health Education in Kansas* until 1952.



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Encephalomyelitis

The Problem It Presents in the United States

DALE W. ANDERSON, M.D., ***Phoenix, Arizona***

SUMMARY

It has been pointed out that the arthropod-borne encephalitides are important because of increasing public interest, lack of trained personnel to control local epidemics, and an epidemiology which is not clearly understood. The encephalitides may become more prevalent because of increased vector population stemming from development of agricultural irrigation projects in endemic areas. Clinical aspects of the problem such as diagnosis, treatment, and prognosis have been discussed with emphasis on methods by which the clinician and specialized personnel may cooperate with one another. The clinician can cooperate best by reporting his patients and making available material for immunological studies. This enables the specialist to enlighten physicians as to the epidemiology and future control of the encephalitides.

INTRODUCTION

It is only recently that physicians have become interested in encephalitides as important disease entities. An analysis of factual knowledge concerning these diseases seems pertinent at present. Even though viral encephalitides have world wide distribution, the disease occurs with sufficient frequency in this country to limit the discussion to various aspects of encephalitis in the United States.

If one is to review encephalitis, certain basic premises must be understood. First, encephalitis is a descriptive term denoting a pathological or clinical syndrome which is unrelated to etiology. It is known that a number of infectious, toxic and allergenic agents are capable of producing an inflammation of the brain, i.e., encephalitis. In this report, the discussion will be limited to common infectious encephalitides defined as "Arthropod-borne virus encephalitides." The encephalitides arising secondarily from infections such as mumps, measles, vaccinia, and influenza, or resulting from toxins and allergens, will not be discussed. Poliomyelitis will be considered only from the viewpoint of differential diagnosis.

HISTORY

Reports of epidemics of encephalitis have appeared since 1871. In 1934, Hayaski isolated a viral agent during one of these epidemics. The disease entity he was studying is now known as Japanese B. encephalitis.[1]

In 1917 and 1918, an epidemic in Australia brought the problem to world notice when J. B. Cleland and collaborators reported a disease entity called Australian "X" disease and proved an arthropod-borne neurotropic virus to be its cause. This disease is now known as Murray Valley encephalitis because of its prominence in this location, and it has been shown to be closely related to Japanese B Encephalitis.[2] In 1930, Meyer, Hering and Howitt, in the Central Valley of California, isolated a virus from the brain tissue of horses with encephalitis. Two years later Meyer reported the possibility that this virus might also be causing disease in humans.[3] Finally, in 1938, the virus causing the disease known as western equine encephalitis was recovered from human tissue by Howitt.[4]

In 1932 an epidemic of encephalitis was reported in Paris, Illinois. It reappeared in 1933 in St. Louis and Kansas City, Missouri. At this time Muckenfus, Armstrong and McCordock isolated the virus of St. Louis encephalitis. In 1933 Ten Broeck and Merrill recognized encephalitis in horses in the eastern states, and in the same year Sittner and Shakan isolated the virus from horses.[1] Then in 1938 Fothergill and collaborators[5] first isolated the virus of eastern equine encephalitis from a human patient, and soon afterwards Webster and Wright[6] isolated the virus from another human being.

Simultaneously with these discoveries, other arthropod-borne viruses were incriminated as causative agents of encephalitis in many parts of the world. At present, almost every geographical area has a particular viral disease entity such as Venezuelan equine encephalitis, Russian far eastern encephalitis, west Nile encephalitis, and louping ill in the British Isles. Only eastern equine, western equine, and St. Louis

This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Anderson is now serving his internship at Good Samaritan Hospital, Phoenix, Arizona.

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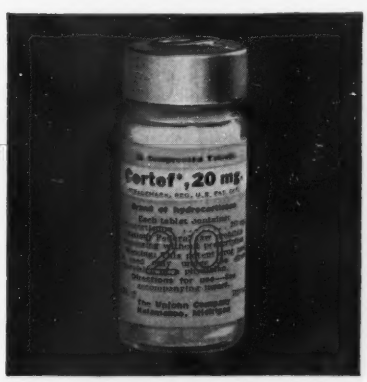

encephalitis are of major importance in the United States.[7]

CLINICAL PICTURE

In the encephalitides, there are no signs or symptoms which enable one to distinguish between various types. At the onset, many of the signs and symptoms are systemic in character and mimic those commonly associated with an upper respiratory infection. Therefore, only in those geographical areas during the season when the disease is endemic or epidemic does the clinical picture have definite meaning to the clinician first seeing the patient.[8]

Cases may be divided into clinical and subclinical entities. Clinical cases are either abortive, mild, or severe. The disease progresses through various phases: prodromal, acute febrile, and convalescent. The onset may be violent or insidious. The incubation period is estimated to vary between 4 and 21 days. Initially, during the first four days, the only symptoms may be headache, tinnitus, malaise, gastrointestinal disturbances, drowsiness, and mild fever.[1] Additional symptoms which may appear the third or fourth day are muscle pains, conjunctivitis, photophobia and nuchal rigidity.[9] At this time restlessness and irritability are seen commonly in children.[10]

An abortive case may occur in which the above symptoms are the only ones to be detected. In such an instance the clinician could only speculate as to the cause. However, the disease process may progress to the acute febrile phase which lasts from one to three weeks and consists of increasingly high fever ranging from 101° to 106° F. with development of a fluxuant, changing neurological picture. Prominent symptoms include severe headache, lethargy, vertigo, and mental confusion.

Among the neurological signs which are extremely variable are tremors, stiff back, diplopia, reflex changes, paralysis, delirium, convulsions, stupor, and coma.[11] In one study involuntary tremors, particularly in the hands in adults, occurred in 50 per cent of the cases.[8] Paralysis occurs only in approximately 15 per cent of the cases.[1] Convulsions which are common in children are usually of the grand mal type. However, in adults, the Jacksonian type of convulsion predominates.[11] In infants one may see a tense fontanelle and cyanosis.[10] The relative frequency with which these signs and symptoms may occur is seen in Table I.[8]

LABORATORY FINDINGS

In encephalitis, positive routine laboratory data are obtained by studying patients' blood and spinal fluid. The findings give no indication as to the type of encephalitis or its severity.[11] Commonly, there is an increase in the white blood cell count to a level varying from 10,000 to 16,000 per cu. mm. in which 70 to 90 per cent are polymorphonuclear leucocytes. Lymphocytes may increase in the later stages of the disease.[12] In one study, there was indication that a lymphocytic rise does not occur readily in individuals under two years of age.[11] In 1938, during an epidemic of eastern equine encephalitis in Massachusetts, white cell counts ranged from 14,600 to 65,900, but all patients with cell counts above 35,000 were found to have pertussis.[10]

TABLE I

The Incidence of Signs and Symptoms in 66 Cases of Encephalitis in Central Valley, California During 1945, 1946, 1947.[8]

Signs and Symptoms	*Frequency (No. Cases)*
Fever	66
Headache	51
Nuchal Rigidity	47
Lethargy or Drowsiness	41
Nausea and Vomiting	38
Stiff Back	31
Tremors	30
Restlessness	28
Reflex Changes	26
Mental Confusion	21
Convulsions	18
Stupor	12
Paralysis (partial)	12
Diplopia	9
Delirium	7
Vertigo	6
Coma	6
Dysarthria	2
Urinary Retention	1

Spinal fluid studies are helpful to the clinician. The spinal fluid may have a "ground glass" appearance with an increased white cell count within the first two days of the illness in 90 per cent of the cases. The number of cells may vary from 25 to 1,000 per cu. mm. Counts over 200 are rare, and counts less than 100 are found in the majority of cases.[8] Polymorphonuclear leucocytes usually predominate, varying from 50 to 75 per cent with a later increase in lymphocytes the second or third weeks of illness.[12] No red blood cells are seen. Initially the total protein can be normal or slightly elevated. Most of the time there is a gradual increase of protein which does not exceed 100 mg. per 100 cc., the usual being 60 to 70 mg. per 100 cc.[8] The globulin fraction is increased four plus.[12]

In a report of 42 cases of western equine encephalitis, Cohen et al. found the protein to be of normal concentration in 34 per cent and elevated in 66 per

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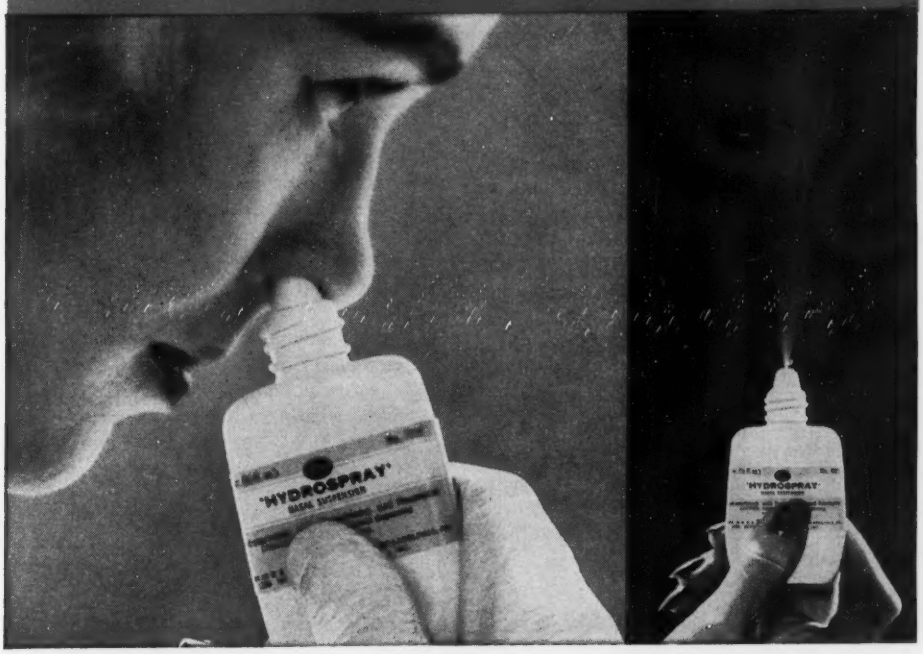

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REFERENCE: 1. Silcox, L. E., *A.M.A. Arch. Otolaryng.* 60:431, Oct. 1954.

cent of the cases.[11] Other spinal fluid findings such as sugar and a chloride determination and specific syphilitic tests will be normal. Colloidal gold may show minor abnormality if the protein content of the spinal fluid is high, but this is not a common finding.[8]

PATHOLOGICAL PICTURE

As in all viral diseases, the basic lesion appears in the infected cell. In encephalitis this cell is the neuron. The areas of the central nervous system in which lesions are most commonly found are thalamus, striate body nuclei, Purkinje and molecular cells of the cerebellar cortex, and the cerebral cortex.

In response to neuronal injury and death, microglia collect around the neurons forming microglial nests which can be seen upon microscopic examination. When damage is severe, these nests may coalesce and appear grossly as sharply demarcated spongy amorphous lesions of necrosing cellular material. Perivascular infiltration by lymphocytes is seen also. Calcium deposition in the brain parenchyma and blood vessel walls has been described in chronic cases, but this is rare.[13]

DIFFERENTIAL DIAGNOSIS

Differentiation among the various types of encephalitis is difficult and among the arthropod-borne encephalitides can be made only by immunological means. This requires long, specialized techniques not readily available to every clinician, so they are as yet of no practical therapeutic value.[8] However, these techniques are of practical value in making a differential diagnosis. Meyer suggested cases of mild encephalitis might be diagnosed and reported inaccurately as non-paralytic poliomyelitis.[12] Headaches, fever, nuchal rigidity, and similar seasonal incidence occur in both poliomyelitis and the primary encephalitides. Lethargy or drowsiness are seen more often in the primary encephalitides, while lower motor neuron weakness with absent reflexes is seen more frequently in poliomyelitis. However, it must be remembered that in any deeply lethargic or comatose patient, reflexes will be absent. In general, absence of involvement of higher centers in the nervous system and predominance of spinal cord signs favor the diagnosis of poliomyelitis.[8]

Distinguishing between arthropod-borne encephalitides and encephalitides secondary to other infections rests solely on the presence or absence of a history of previous exanthem, vaccination, or on immunological studies. Secondary encephalitides as a rule occur in late fall, winter, or early spring months, while arthropod-borne encephalitides occur in late spring, summer, and early fall months.[8]

IMMUNOLOGY

The most important aspects in establishing a specific etiology in arthropod-borne encephalitides are concerned with serological studies on the patients' serum.[8] Various attempts in isolation of the virus from blood, feces, and nervous tissue have been successful, but these types of studies are too detailed for routine use.[14] The two serological studies which are used mainly are the complement fixation and the neutralization tests. The techniques of these tests are complicated, but in principle they are relatively simple.

The complement fixation test is based on the ability of a specific antigen-antibody reaction to fix known complement. When sheep red blood cells and specific antibody for these cells are added, hemolysis will result unless the complement has been removed by the previous antigen-antibody reaction.[15]

The neutralization test depends on the ability of a specific antibody to neutralize a virus when inoculated simultaneously into a susceptible host. Quantitative results are obtained by titrating known sera against unknown sera.[16] These specific serological tests now are possible for eastern equine, western equine, St. Louis, Japanese B, Venezuelan equine, Russian tick-borne encephalitis and other encephalitides of viral etiology.[8]

It is important for the clinician to know how and when to obtain blood specimens for these tests. Positive diagnosis of active infection in a patient requires the demonstration of a significant rise in antibody titer in at least two blood samples. The first specimen should be drawn as nearly as possible after onset of the illness. The second specimen should be obtained from 7 to 21 days later, depending on duration of the illness and rate of recovery. If the duration of illness is short, the second specimen should be drawn seven days later; if the illness is long, the second specimen should be obtained at the height of the illness. The specimens should consist of 10 cc. sterile quantities of whole blood which should be kept at non-freezing refrigeration until shipment to the laboratory. Blood samples should always be drawn in suspected cases because of the importance for epidemiological studies and prevention of encephalitides, even though no practical therapeutic aid is gained from them.[8]

Immunological studies have revealed differences in antibody responses among the specific encephalitides. Both neutralizing and complement fixing antibodies begin to develop at the onset of the disease and reach a high titer at the end of the first week in western and eastern equine encephalitides.[1] In St. Louis encephalitis, the antibodies rise much more slowly and do not reach high titers for several weeks.[12] In Japanese B encephalitis, neutralizing

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antibodies appear from the third to 14th day of illness while complement fixing antibodies appear from the fifth day to the fifth month. There is some indication that neutralizing antibodies are present longer than complement fixing antibodies; the former may last from four years to life, the latter for only one to three years.[1] It is of interest that patients with Japanese B encephalitis may show an anamnestic response. Patients having only neutralizing antibodies present will show a considerable rise in complement fixing antibodies after a minimal stimulating dose of Japanese B vaccine.[17]

As an example of the results of the immunological studies on a series of suspected encephalitis patients, a report of 90 such cases in Kern County, California, occurring between June to September of 1952, is cited. Among the 90 cases studied, 42 had positive tests for western equine encephalitis, 4 for St. Louis, 9 for mumps encephalitis, and 35 were negative to all tests. The patients within the latter group were diagnosed as having either non-paralytic poliomyelitis or were presumed to have had some type of arthropod-borne encephalitis.[11] The percentage of positive results in the antibody studies reported by Howitt varied from 26 to 95.[18] Negative tests are still to be explained in patients with clinical encephalitis. One could postulate that other viruses, not isolated yet, are the cause of illness in such patients. It is the author's suggestion that a virus related to the psittacosis-lympho granuloma venereum group could be incriminated in the above cases. Such a virus has been found as the etiologic agent of 16 epidemics of bovine encephalitis from 1948 to 1952 in the western United States.[19]

STATISTICS

Analysis of the reported vital statistics in the encephalitides can give the physician an idea as to the prognosis of his patient. The incidence of disease with reference to age and sex of the patients depends on environmental factors affecting exposure. There is no characteristic age or sex incidence for any one of these diseases; therefore, the greatest insight can be gained by analysis of the classical epidemics reported for each encephalitis. In general it can be stated that variation in fatality is partly dependent on age incidence, being greater in the higher age groups.[20]

In western equine encephalitis, the majority of patients are males, 20 to 50 years of age, who work outdoors.[1] In recent small sporadic epidemics, the highest incidence has been in infancy, the lowest in the 5- to 12-year group, and a moderately high incidence in the 15- to 50-year group. In Pineal County, Arizona, in 1941 an epidemic consisting of 18 cases of St. Louis and western equine encephalitis was reported in which 50 per cent of the patients were under 1.2 or 1 to 2 years of age.[9]

The mortality rate in epidemics of western and St. Louis encephalitis appears to be between 5 to 20 per cent in the mid-west.[21] A severe epidemic of western equine encephalitis reported in 1941 in North Dakota, Minnesota, and neighboring Canada, consisting of approximately 3,000 cases, had a mortality rate of 8 to 15 per cent.[22] In a California epidemic of St. Louis and western equine encephalitis in 1952, consisting of 727 cases, 51 deaths were reported.[23]

Epidemics of eastern equine encephalitis have occurred infrequently. The first reported epidemic occurred in eastern Massachusetts in 1938 with a total of 39 cases in which an over all mortality of 65 per cent was seen. Sixty-five per cent of the cases were in patients under 10 years of age with equal sex distribution.[24] A sporadic epidemic occurred in southern Louisiana in 1947 with a 70 per cent mortality rate. Of the 10 cases reported, 7 were in patients under 10 years of age. In 1951, an epidemic of 13 cases occurred in the Yaque River Basin in Canada. The majority of cases were in patients under 5 years of age with 9 deaths reported.[25]

Statistics reported from countries other than the United States have been similar. Japanese B encephalitis in recent years is reported to have a mortality rate of approximately 28 to 35 per cent with the greatest age incidence in the 5- to 9-year old groups. Murray Valley encephalitis in Australia has a mortality rate of approximately 70 per cent, and the greatest incidence is in the 0 to 5-year group.[1] Russian Far Eastern encephalitis has a mortality rate of 30 per cent.[26]

Buss and Eaton made a report of mortality rates of poliomyelitis and the encephalitides in Kern County, California, between 1941 to 1950. Their report showed that the mortality rates are similar, but the number of cases of poliomyelitis was greater than those of encephalitides. The great difference between poliomyelitis and the encephalitides lies in the sequelae or neurological residuals resulting from such infections. In poliomyelitis, sequelae consist of partial or complete paralysis of the lower motor neuron type, while those of the encephalitides consist of upper motor neuron deficit. Sequelae in the encephalitides appear in 10 to 40 per cent of cases, depending on the age group involved. Very young patients have more sequelae than those in older age groups.[1] Residuals consist of emotional instability, mental retardation, hydrocephalus, epileptiform seizures, tremors, weakness, Parkinsonism, and frank paralysis.[9] Decerebrate rigidity has been reported.[17] As in poliomyelitis, these sequelae are the most feared result of such infections.

TREATMENT

Specific treatment for the encephalitides has not been forthcoming as have antibiotics for bacterial

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infections. Therefore, therapy is entirely symptomatic. Salicylates should be used freely for control of temperature elevations.[11] There is no specific indication for the use of antibiotics or sulfonamides, because at present, no antibiotic has been shown effective against viruses. These drugs should be used to control intercurrent infections. Convulsions can be controlled by the usual anti-convulsive drugs. Phenobarbital in one-half to one grain doses, three or four times a day, adding 32 to 65 mg. of dilantin one to four times a day as indicated, is usually adequate for control. The lethargic or comatose patient will require around the clock nursing care.[8]

Muscle spasm or spasticity can be treated with hot packs or a modified Kenny treatment. Spastic paralysis requires active and passive physiotherapy in severe cases.[12]

Maintenance therapy is of extreme importance. The physician should pay special attention to proper fluid balance, caloric intake, care of incontinence with indwelling catheters, and avoidance of decubitus ulcers by frequent movement of the patient.[8] Ascorbic acid, even in large dosages, has been of little value.[11]

Recently, because the utilization of ACTH and cortisone has produced some beneficial effects in acute allergic disseminated encephalitis, these hormones have been tried in the viral encephalitides.[27] Clinical and experimental evidence pointing to the fact that fatigue, chilling, or pregnancy lowers resistance and increases paralysis in poliomyelitis, has led to the thought that the stress reaction may play a part in the viral encephalitides. This has not proved to be the case, however, since both experimental and clinical studies with these drugs have produced negative results.[28]

Immune serum has been given clinical trials, but it has proved valueless if it is begun after the onset of definite clinical symptoms. Passive immunization with specific anti-serum is being used experimentally in animals but not as yet in humans.[12] Active immunization with a formalinized inactivated virus is now possible. For several years veterinarians have used a formalinized chick embryo vaccine in horses, approved by the United States Bureau of Animal Industry.[12] Vaccination in horses has not appeared to reduce the disease in man.[20]

Although vaccines are available commercially for eastern equine, western equine, and St. Louis encephalitis, at present they are used only for people heavily exposed to these agents in the laboratory.[20] Public use is impractical because of cost, temporary immunity, and relatively low clinical attack rate as compared with inapparent infection rate.[7] Practical vaccination does seem promising because Hammon has isolated a strain of virus which has the antigenic characteristics of western equine, eastern equine *and* St. Louis encephalitis viruses. From this finding he postulates that there is a stem virus from which these three viruses arose, and if the stem virus is found, this virus could be utilized for preparation of a vaccine for the three most prevalent encephalitides in the United States.[29]

EPIDEMIOLOGY

All the arthropod-borne encephalitides have been found to have a common epidemiological pattern (not identical). The virus is transmitted to humans through an arthropod vector such as the mosquito. Ticks and mites have also been shown to be vectors. There is a correlation between the number of human cases, positive mosquito pools, and equine cases.[8] Nearly every severe epidemic in humans has been preceded by an epidemic in horses in the same area. It is thought that the natural epidemiological cycle for these diseases is as follows:

Humans and equines enter the cycle accidentally.

The geographical distribution of the three viral encephalitides in the United States differs somewhat. Western equine encephalitis appears in all states west of the Mississippi River, as far east as Michigan in the north, and Alabama and Florida in the south. The virus has not been found in an area when it has not been present in the equine population also. St. Louis encephalitis is known to be present in the Pacific Coast area, the southwestern, and midwestern states. Our knowledge of its distribution is limited because of the lack of equine infection. Eastern equine encephalitis is distributed along the eastern seaboard to New England in the north, Florida in the south, and as far west as Michigan in the north and Texas in the south. Again, equines are helpful in the study of the virus.[7] This is the only virus of the three reported outside the

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Temperate Zone.[30] Both western equine and St. Louis viruses are commonly found in the same states, but only four states harbor all three viruses: Texas, Alabama, Michigan, and Arkansas.[31]

Several features are common to all areas where the encephalitides are present. All have high summer temperatures with moderate temperatures in the winter and springtime.[23] Seasonal rains are heavy with intermittent flooding, or irrigation is common. Large numbers of mosquitoes are present at least every few years.[20] Epidemics appear when there are increased demands for irrigation or when heavy rainy seasons increase the vector population.[8]

The greatest incidence of infection is seen in small towns and rural and suburban areas while the centers of large cities are relatively free of these infections.[20] In antibody studies to detect subclinical infections, the highest incidence of antibodies is found near towns, with decreasing numbers of positive sera being found as one moves away from the towns.[25] It was also noted that the number of positive serums among normal individuals increased with extended residency in the endemic areas but not with age.[20] Positive sera for more than one virus and poliomyelitis were obtained in numerous persons.[18]

Vectors for the encephalitides consist of blood-sucking arthropods. Three genera of mosquitoes, Culex, Aedes, and Culiseta, have been incriminated. Culex is the genus considered most important in the United States.[7] Bird parasites such as ticks, mites, and lice harbor the virus. Although mites have not been studied extensively, they are interesting in that the virus is transmitted trans-ovarially in chicken mites.[30]

Much energy has been expended in the search for a natural reservoir host for the viruses. During the search many wild and domestic fowls and mammals have been incriminated by antibody studies. Among animals shown to have antibodies present are chickens, ducks, doves, pigeons, English sparrows, cowbirds, blackbirds, pheasants, squirrels, rabbits, and horses. From this evidence, it appears that birds are the most important vertebrate reservoirs for viral agents causing encephalitis. It is of interest to note the low incidence of antibodies in animals studied in the midwest, as opposed to other areas.[30] This may be an indication that the true reservoir host in the midwest has not been found.[32]

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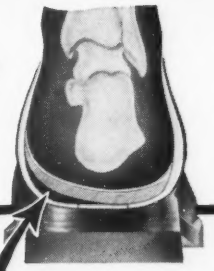

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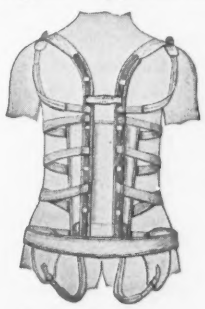

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Chlordane Poisoning

The Committee on Pesticides of the A.M.A. has issued a warning on the danger of poisoning by chlordane, an agricultural and household insecticide used to combat grasshoppers, ants, flies, mosquitoes, and roaches. Poisoning may be caused by repeated skin contact, breathing of fumes, or accidental swallowing.

The onset of symptoms is influenced by the means of absorption. Acute signs usually appear within 45 minutes after swallowing the poison, and death may occur within 24 hours.

Treatment consists of removal of the poison from the stomach or skin, followed by a salty purge and administration of sedatives. In case of skin contact, the contaminated area should be washed immediately with soap and water. Fatty substances such as milk or oil purgatives speed absorption of the poison and should be avoided.

Booklet on Indigent Care

Current information on 18 representative state and local indigent care plans is included in a booklet recently published by the A.M.A.'s Council on Medical Service. The material included was gathered from a study which has been continuing since 1952. Copies may be secured on request.

State plans outlined are those of Rhode Island, New York, Pennsylvania, Maryland, Illinois, Washington, and North Carolina. The plan used in Topeka is described in the section devoted to local programs.

Most types of cancer show a greater incidence among men than women, which may result from a different degree of exposure to environmental factors, according to data from metropolitan areas studied by the National Cancer Institute. The difference in incidence rates increases with age, especially for respiratory cancer, leukemia, and cancer of the buccal cavity.

Only cancer of the breast, reproductive organs, and certain endocrine glands was found to occur more frequently among women.

The report notes a positive correlation between cancer incidence and chronological age—the older the person the greater the likelihood of cancer. Half the people with diagnosed cancer, both men and women, were between 50 and 70 years of age.

Men appear more susceptible to cancer in the first two and the last two or three decades of the usual lifetime, whereas women have a higher rate during the childbearing years. In fact, at about age 35, relatively twice as many women as men are found to have a malignant tumor. After the childbearing period, however, the male rate catches up with and exceeds the female rate.

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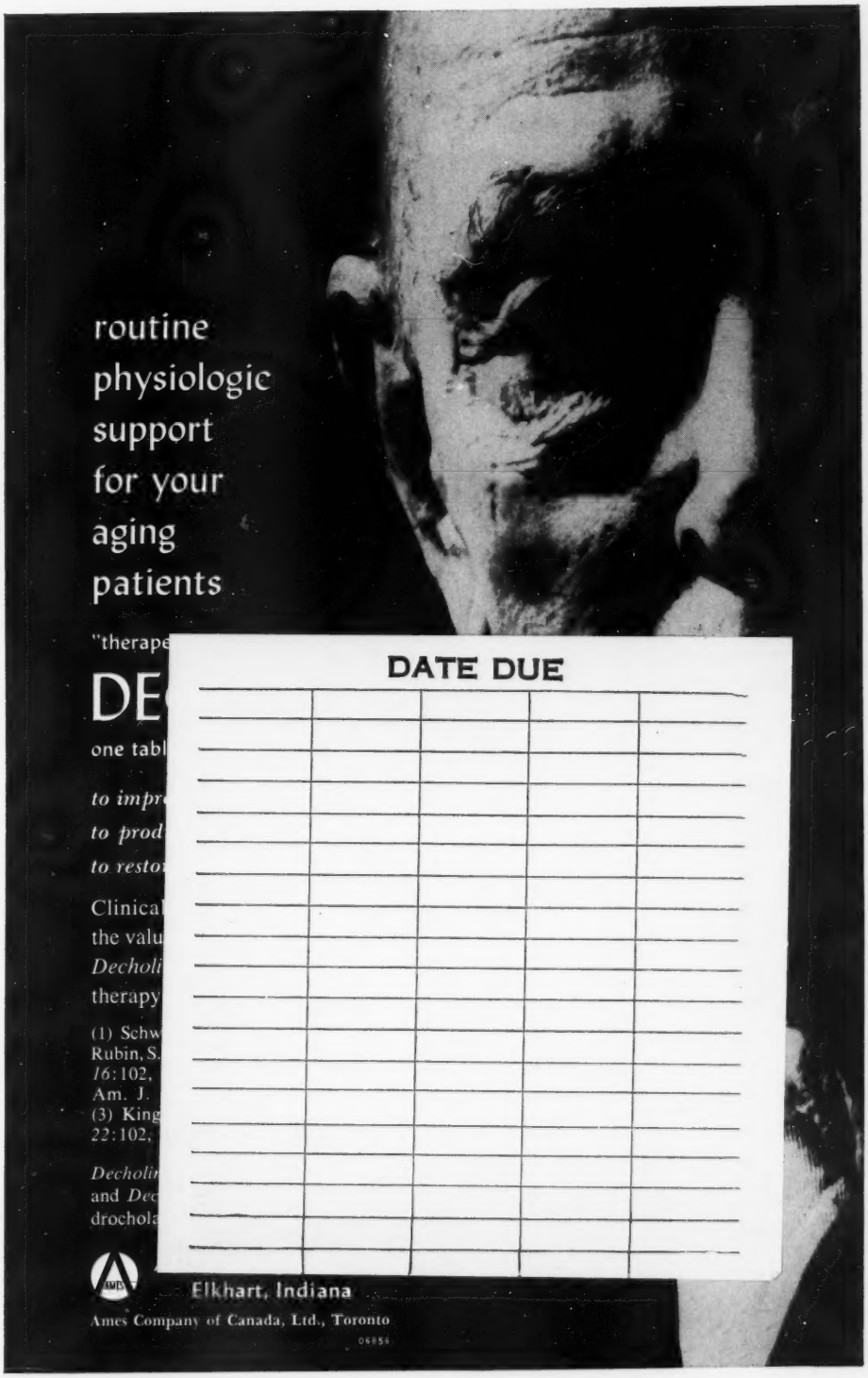
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